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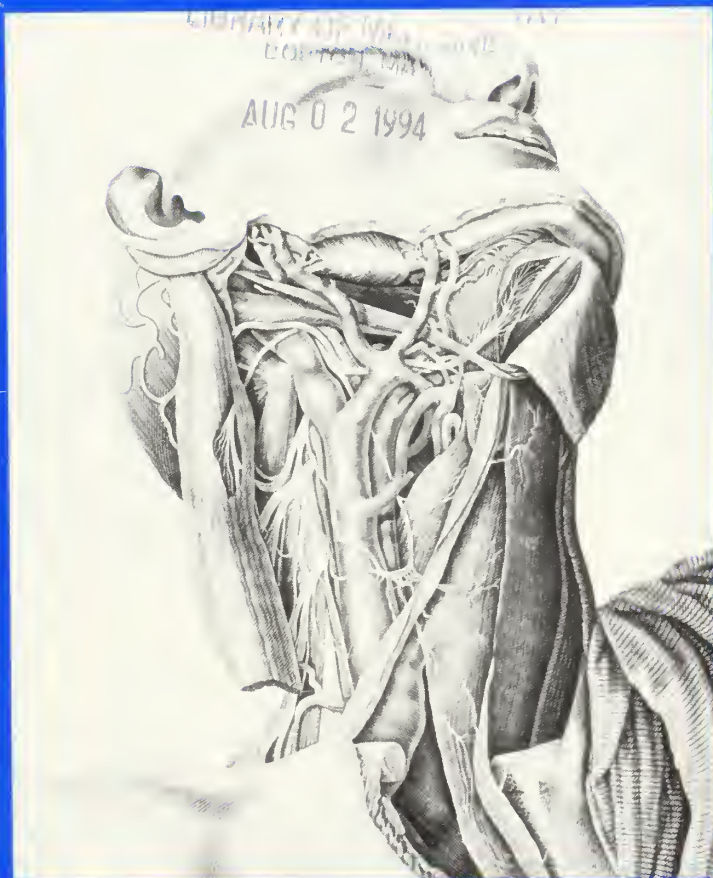


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**SPECIAL ISSUE: CEREBROVASCULAR  
DISEASES, PART 2**  
**GUEST EDITOR: ARTUR PACULT, M. D.**

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# President's Page

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## THE NEW DOCTOR-INSURANCE COMPANY-PATIENT RELATIONSHIP CONTRACT

I have become aware of two recent instances involving South Carolina physicians and health insurance company managed care contracts that can serve as "lessons learned" for the rest of us. Each of these problems is different and yet the solutions are similar. In previous remarks we have discussed the necessity of partnerships, risk sharing and written negotiated agreements in order to succeed in this new health care environment. In fact, mutual expectations and commitments are more easily remembered and honored if clearly documented and shared. Contracts can help avoid misunderstandings, promote better patient care, contain costs and make things easier for providers, recipients and payors. Unpleasant surprises can best be avoided by patients and physicians if we do our homework and ask the right questions before signing managed care agreements.

The first of these current situations came to the attention of the SCMA staff after an advertisement appeared in one of the state's large daily newspapers offering a new managed care product with several hospitals and hundreds of physicians. Our members began calling the SCMA office to inquire as to the source and affiliation of this physician brigade. A telephone call to the insurance carrier involved in this network revealed the answer. It seems that in their preferred provider contracts with SC doctors they specify that the enrolled physician may be assigned to future managed care plans entered into by this company. A number of our callers were not aware that they were included in the doctors participating in this new product. The insurance carrier had done nothing awry. All of the prior contracts clearly stated the company's intent to include them in future business. Our colleagues either did not read the documents carefully enough prior to signing or they did not remember the specifics.

The other contract snafu was related to me by a business person in the upstate. She had developed an excellent relationship with a young physician who had been in practice for almost two years. Her doctor then decided to join a larger group for stability, cost and call sharing. When she approached the reception desk at her physician's new office to make her first appointment, she was told that her health insurance carrier did not have a contract with their doctors and the cost of her care would not be reimbursed. This lady's health insurer's contract prevented her from continuing the relationship with her chosen physician. After obtaining a list of the providers who were still affiliated with her managed care plan, she was even more perplexed. There was not a single familiar name and the office addresses were all on the other side of town. As she reviewed her insurance papers, she found there was no real "point of service" option for her to pursue. The lesson to be learned is that our patients must become well-informed consumers of medical services and develop expertise in interpreting health care contracts and agreements. Both physicians and patients will need to master the skills to negotiate unacceptable items in these documents prior to signing.

Ambrose Bierce once said that "absurdity is a statement or belief manifestly inconsistent with one's own opinion." Many of us feel, and rightfully so, that it is absurd for the inherently simple relationship between a doctor and his/her patients to be subject to so much interference. These contracts and complexities are manifestly inconsistent with opinions of doctors and patients, but they are a reality of the modern health care environment. Caveat Emptor.

*O. Marion Burton MD*

O. Marion Burton, MD  
*President*

## Contents

### Special Issue: Cerebrovascular Diseases, Part 2

Guest Editor: Artur Pacult, M. D.

#### 309 Introduction

Artur Pacult, M. D.

#### 310 Clinical Syndromes of Cerebral Ischemia

Timothy D. Carter, M. D., Jerome E. Kurent, M. D.

#### 314 Medical Imaging of Cerebral Ischemia

Michael C. Garovich, M. D.

#### 318 Color Flow Duplex Scanning in Evaluation of Carotid Artery Atherosclerotic Occlusive Disease

J. E. Fogartie, Jr., M. D., R. V. T., W. O. Holloway, M. D., J. R. Hobson, Jr., M. D., Jacqueline Davis, R. V. T., Mary Camp, R. V. T., Peggy Brown

#### 327 The Surgery of Extracranial Vascular Occlusive Disease

Edward C. Morrison, M. D.

#### 330 Medical Management of Ischemic Cerebrovascular Disease

John W. Plyler, M. D.

### Special Article

#### 341 Report of the SCMA Delegation to the AMA Annual Meeting

Walter J. Roberts, Jr., M. D.

### Editorials

#### 339 The Magnitude of the Problem

Artur Pacult, M. D.

### Features

#### 347 Alliance Page

#### 345 On the Cover

#### 307 President's Page

### Association

#### 349 Gray Matter

#### 323 SCMA Newsletter

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## SPECIAL ISSUE: CEREBROVASCULAR DISEASES, PART 2: INTRODUCTION

GUEST EDITOR: ARTUR PACULT, M. D.\*

In January of this year, we presented our colleagues with Part I of *The Journal's* special issue on cerebrovascular diseases, devoted to problems affecting the brain, by its tendency to hemorrhage. Symptoms, diagnosis and current therapy of different types of aneurysms and vascular malformations were discussed.

In this issue of *The Journal*, several authors concentrated on the type of ischemic diseases of the cerebral vessels. In doing so, Drs. Timothy Carter and Jerome Kurent discuss clinical syndromes of cerebral ischemia. Dr. Jim Fogartie has written on the use of color flow Duplex scanning in evaluation of carotid occlusive disease. There are articles by Dr. Michael Garovich on current medical imaging

techniques of cerebral ischemia and by Dr. Ed Morrison on the subject of surgery of extracranial vascular occlusive disease. Finally, Dr. John Plyler discusses medical management of ischemic cerebral problems.

The terms "apoplexia cerebri," "insultus," or "ictus cerebri," were known since many centuries ago, but only in XVII century, after autopsy studies by Wepfer, it was recognized that the etiology of the stroke is a damage to the cerebral vessels.

We hope that the reader will find this, as well as the previous issue on cerebrovascular diseases, interesting and helpful in our everyday practice of medicine. □

---

\*125 Doughty Street, Suite 400, Charleston, SC 29403.

# CLINICAL SYNDROMES OF CEREBRAL ISCHEMIA\*

TIMOTHY D. CARTER, M. D.  
JEROME E. KURENT, M. D.

---

Stroke is the third leading cause of death and a leading cause of adult disability in the United States. There are approximately 500,000 new stroke patients yearly with a 25 to 30 percent death rate.<sup>1</sup> As promising new therapies are developed, increasing emphasis will be placed on the clinical diagnosis of transient cerebral ischemia and stroke so that patients can be optimally managed.

## GENERAL PRINCIPLES

In the broadest sense, stroke is caused by one of two different processes – ischemia or hemorrhage. Stroke of either type is usually characterized by the sudden onset of neurologic dysfunction. A neurologic deficit evolving slowly over weeks to months is not likely to be secondary to a stroke. While hemorrhagic events frequently have a catastrophic presentation, the symptoms of cerebral ischemia may be mild or so subtle that their significance is misinterpreted by both patient and physician. Transient ischemic attacks (TIAs) are episodes of neurologic dysfunction secondary to cerebral ischemia which resolve within twenty-four hours. More commonly, TIAs last only a few minutes and are frequently dismissed by patients as no cause for alarm. However, TIAs represent a significant risk factor for stroke, and their recognition may allow intervention by the physician in order to prevent permanent ischemic cerebral injury. Thus, any patient who complains of transient visual loss (amaurosis fugax) speech disturbance, numbness, focal weakness, dizziness, vertigo, or other transient neurologic symp-

toms warrants careful evaluation.

Several different pathophysiologic processes may lead to ischemic stroke. Thrombosis of large or small arteries is the most common cause of ischemic stroke. Underlying cerebrovascular atherosclerosis frequently leads to thrombosis and may provide a source of emboli to distal cerebral arteries.<sup>1</sup> Large artery thrombotic stroke is preceded by TIAs in forty percent of patients.<sup>2</sup> The onset of thrombotic stroke is often during sleep and the course frequently fluctuates.<sup>2</sup> In contrast, stroke secondary to cardiogenic embolism usually occurs suddenly during physical activity. Systemic hypoperfusion may also lead to ischemic stroke but the cause is usually obvious (hypotension, gastrointestinal bleeding).<sup>2</sup>

## NEUROANATOMIC SYNDROMES

The signs and symptoms of cerebrovascular disease vary with the area of brain affected (see Table 1). Because of the known blood supply and functional anatomy of the brain, disturbance of blood flow to a major cerebral artery usually produces characteristic signs and symptoms. The first differentiation can be made between territories supplied primarily by the carotid arteries (anterior circulation) and territory supplied by the vertebrobasilar system (posterior circulation). Posterior circulation ischemic injury is often manifested by evidence of brain stem dysfunction which most frequently results in "crossed" findings on examination, i.e., finding evidence of cranial nerve dysfunction on one side of the face and head accompanied by motor or sensory deficits on the opposite side of the body. Dizziness, vertigo, diplopia, and dysarthria are common symptoms of vertebrobasilar

---

\*From the Department of Neurology, Medical University of South Carolina, 171 Ashley Avenue, Charleston, SC 29425.



**TABLE 1**  
**SOME COMMON SYNDROMES OF CEREBRAL ISCHEMIA**

<b>I. ANTERIOR CIRCULATION ISCHEMIA</b>	
<i>Signs/Symptoms</i>	<i>Localization/Cause</i>
Amaurosis fugax	Ophthalmic artery occlusion causing retinal ischemia
Pure Motor Hemiparesis	Internal capsule lacunar infarction
Pure hemisensory deficit	Lacunar infarction affecting the posteroventral thalamic nucleus
Hemiparesis (Face, Arm>leg), Hemisensory deficit, homonymous, hemianopsia, aphasia	Dominant (usually left) hemisphere in middle cerebral artery territory
Hemiparesis (Face, arm>leg), hemisensory deficit, homonymous hemianopsia, left sided neglect, visuospatial disturbances	Non-dominant (right) hemisphere in middle cerebral artery territory
<b>II. POSTERIOR CIRCULATION ISCHEMIA</b>	
<i>Signs/Symptoms</i>	<i>Localization/Cause</i>
Vertigo, nystagmus, vomiting, Homer's Syndrome cerebellar signs, hoarseness, loss of face & temp sensation, contralateral loss of pain & temp from body	Lateral medullary infarction secondary to occlusion of posterior inferior cerebellar or vertebral artery
Hemiparesis with contralateral CN III palsy	Medial midbrain infarction
Hemiparesis with contralateral CN VI palsy	Medial pontine infarction
Homonymous hemianopia (especially upper quadrant)	Calcarine cortex or optic radiations

ischemia. Isolated cranial nerve dysfunction due to brainstem ischemia, or ataxia secondary to cerebellar dysfunction commonly occur with posterior ischemia. The "locked-in" syndrome, or de-efferented state, is due to infarction of the ventral pons. These patients are alert, but motor ability may be limited to eye blinking.

Anterior circulation ischemia results in some of the more common stroke syndromes. The anterior cerebral arteries are paired parasagittal vessels which predominantly supply areas of the brain controlling the contralateral leg. Thus, acute lower extremity monoplegia is the most frequent presentation of anterior cerebral artery ischemia.

The middle cerebral artery supplies larger areas of cortex in the frontal, temporal, and parietal lobes. Because of lateralization of hemispheric function, the symptoms of middle

cerebral territory ischemia are different depending on the side affected. Language disturbances such as aphasia are frequently seen with dominant hemisphere dysfunction. The left hemisphere is dominant in right-handed individuals and most left-handed people. Confusion as well as neglect of the left side of the body is typically associated with nondominant right cerebral lesions. Motor and sensory function of the face and arm are subserved by areas of brain supplied by the contralateral middle cerebral artery. Thus, many strokes are localizable to the middle cerebral artery territory by the finding of hemiplegia affecting primarily the face and arm more than the leg. Aphasia will be present if the left hemisphere is involved, or left-sided neglect if the right hemisphere is affected.

Some strokes do not involve major vessels but are secondary to occlusion of smaller pen-

etrating arteries which are especially numerous in the basal ganglia and pons. Lacunar infarcts are small areas of infarction due to thrombosis of these small vessels and are common in patients with hypertension. The clinical manifestations are quite variable depending on specific regions of the brain affected (see Table 2). Pure sensory or motor findings are the most common lacunar syndromes.<sup>3</sup> While the affected area of brain may be rather small, lacunar strokes can result in considerable disability. For example, a small lacunar infarction involving the internal capsule can result in dense hemiplegia affecting the contralateral face, arm and leg.

Intracerebral hemorrhage usually occurs as an acute catastrophic event with severe neurological deficit, coma or death. The mortality from intracerebral hemorrhage is high. Rapid diagnosis made by computed tomography (CT) is critical in order to maximize therapeutic effort.

Subarachnoid hemorrhage (SAH), is most commonly caused by a ruptured berry aneurysm. The patient usually presents with acute onset of severe headache and stiff neck.

Focal neurological deficits, coma and death are common, but patients with grade I SAH may have no focal deficit. The clinician must have a high index of suspicion for this disorder when evaluating any patient with new onset of severe headache. Early diagnosis is vital in order to offer the best chance of survival by neurosurgical clipping of the aneurysm once it has been demonstrated by cerebral angiography.

## PROGNOSTIC AND THERAPEUTIC IMPLICATIONS

Anatomic syndromes of cerebral ischemia may provide prognostic information for the clinician. For example, a recent study demonstrated that patients suffering from large anterior circulation infarcts having both cortical and subcortical involvement had a "negligible chance of good functional outcome" and high mortality while those with posterior circulation infarcts had the best chance of a good outcome with 62 percent of these patients becoming functionally independent.<sup>4</sup>

Even in an era of sophisticated neurodiagnostic testing, the clinical diagnosis of cere-

**TABLE 2**  
**LACUNAR SYNDROMES**

1. Pure sensory stroke or TIAs
2. Pure motor hemiparesis (PMH)
3. PMH variants"
  - modified PMH with "motor aphasia"
  - PMH sparing face
  - PMH with various extraocular movement palsies
  - PMH with confusion
4. Ataxic hemiparesis
5. Dysarthria – clumsy hand syndrome
6. Mesencephalothalamic syndrome
7. Thalamic dementia
8. Cerebellar ataxia with crossed third nerve palsy
9. Sensorimotor stroke (thalamocapsular)
10. Hemiballism
11. Lower basilar branch syndrome
12. Lateral pontomedullary syndrome
13. Lateral medullary syndrome
14. Locked-in syndrome (bilateral PMH)
15. Sudden onset loss of memory
16. Weakness of one leg with ease of falling
17. Pure dysarthria
18. Acute dystonia of thalamic origin

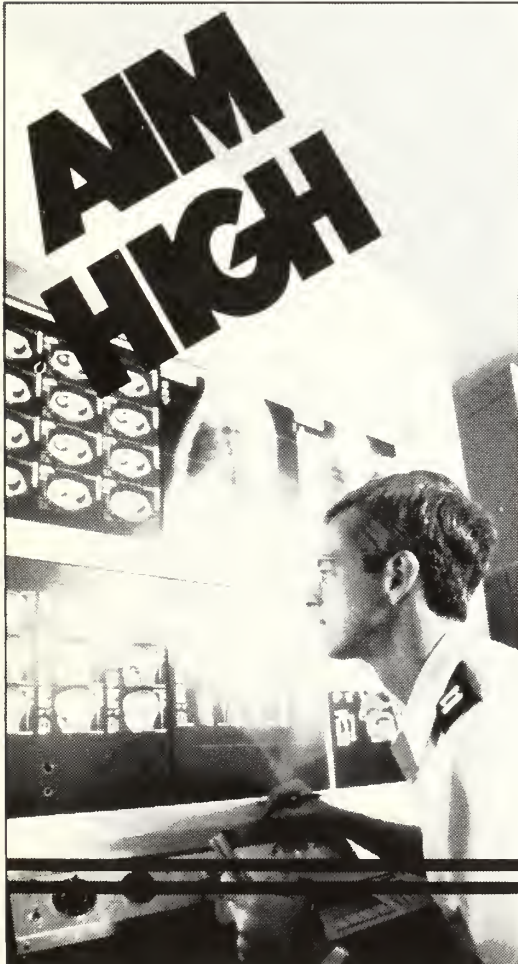
*Adapted from Fisher<sup>3</sup>*

bral ischemia is of critical importance. Computed tomography (CT) or magnetic resonance imaging (MRI) are often normal in the first hours after an acute ischemic event. Management of the stroke patient must be guided by clinical judgement. Localization of the problem to the appropriate vascular territory can help to guide subsequent evaluation of the stroke patient. For example, evaluation of the carotid arteries in a patient experiencing brain stem TIAs will likely be unrewarding. Conversely, the patient with transient bouts of aphasia and right arm weakness (left middle cerebral artery territory) may benefit from endarterectomy if there is high grade stenosis of the left carotid artery. Appropriate medical therapy may also be indicated by the clinical syndrome. For example, a patient with atrial fibrillation who has made a good recovery

from a right middle cerebral artery territory infarction would probably benefit from long term anticoagulation with warfarin. However, a patient recovering from a lacunar infarction would be more appropriately treated by long term control of hypertension and perhaps with agents which modify platelet function such as aspirin. □

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# MEDICAL IMAGING OF CEREBRAL ISCHEMIA\*

MICHAEL C. GAROVICH, M. D.

Cerebrovascular disease is a major cause of morbidity and mortality in the United States affecting 400,000 people annually.<sup>1</sup> The causes are diverse, including thrombosis, embolism, vasospasm, hypoperfusion, coagulopathy and trauma.

This article will focus on the CT, MR, conventional angiography and MR angiography findings of thromboembolic phenomena. It will concentrate on two major clinical divisions:

1. Prestroke states (reversible ischemia and bruits)
2. Stroke (irreversible ischemia).

New advances in drug treatment, neuroprotective agents, thrombolytic therapy and surgery have made the early and precise diagnosis of ischemic cerebrovascular disease paramount.<sup>2</sup>

## BRUITS

Four percent of the population have asymptomatic carotid bruits over the age of 40.<sup>3</sup> These patients are at increased risk for both stroke and myocardial infarction. Emboli from the carotid bifurcation is a principal cause of stroke.<sup>4</sup> Clinical trials have shown that carotid endarterectomy is efficacious in patients with TIAs who have greater than 75 percent stenosis at the bifurcation.<sup>5</sup> The value of endarterectomy for asymptomatic stenosis is unclear, but large trials are underway.<sup>6</sup> The potential number of patients who could benefit from surgical and medical intervention is very large. The importance of non-invasive, accurate, and cost effective medical imaging of the carotid bifurcation is obvious.

Imaging of the carotid bifurcation is typical-

ly done by conventional angiography or non-invasively by MR angiography (MRA) or duplex ultrasonography.

Angiography is the gold standard for imaging the intra-cranial and extracranial cerebral vasculature. Recent technical advances include: (1) less traumatic, smaller catheters (5Fr, 4Fr), (2) less toxic contrast agents, and (3) digital radiography which can decrease catheter time and lower contrast load. These and other technical improvements have decreased the risk of the procedure. Despite these advances, complications do occur. The rate of permanent neurologic defect is 0.15 percent to one percent in large series.<sup>7,8</sup>

Magnetic resonance angiography (MRA) is an exciting new development that has tremendous potential. It can non-invasively image cerebral blood flow without contrast. While conventional angiography depicts the anatomic vessel lumen, MRA is more physiologic depicting flowing blood (Figure 1).

MRA manipulates differences between stat-



Figure 1. Acute left hemispheric infarct. 3D TOF MRA of the circle of willis. The left internal carotid artery demonstrates no flow. The left anterior cerebral and middle cerebral arteries show decreased flow. They fill through the anterior communicating artery and other collateral vessels.

\*From Trident Regional Medical Center, 9330 Medical Plaza Drive, North Charleston, SC 29418.

ic brain tissue and flowing blood to generate contrast and selectively highlight the vasculature. There are two basic techniques: time of flight (TOF) and phase contrast (PC).

TOF utilizes saturation pulses that decrease signal (darken) in stationary tissue. Rapidly flowing blood entering into region is not subjected to the saturation pulse and has relatively increased signal (bright, flow related enhancement).

Phase contrast relies on phase differences between stationary tissue and moving blood. It is a subtraction technique in which static tissue yields no signal and moving blood demonstrates signal proportional to velocity.

3D and 2D methods are used for both techniques. Computer post processing is utilized to create projection images similar to conventional angiography. MRA should be correlated with routine brain MRI images.

MRA has proven efficacious when compared to conventional angiography.<sup>9,10</sup> Patients who have normal or mild stenosis at the bifurcation by MRA generally require no further evaluation. MRA is also quite effective in severe or total occlusion. MRA may overestimate the degree of narrowing with moderate stenosis due to complex flow and intravoxel dephasing. This can be limited by using newer techniques that decrease dephasing and by reviewing the original data set. At present, MRA is best utilized as a screening technique eliminating normal and mild stenoses from further workup. If surgery is contemplated, conventional angiography is recommended to confirm MRA results.

Duplex sonography is also effective in the evaluation of the bifurcation and is less expensive. It is technically demanding with accuracy being sonographer dependent. It is less effective with anatomically high bifurcations and does not evaluate other potential sites of atherosclerosis, e. g., aortic arch, great vessel origin, or paracavernous internal carotid artery.

Conventional angiography, MRA, and duplex sonography are all insensitive to ulceration when compared to surgical specimens.

Conventional angiography is best with a detection rate of 67 percent.<sup>11</sup> No imaging technique can detect microulceration which may be clinically important.

### TIA

CT is insensitive and usually normal with clinically reversible ischemia (TIA, RIND). Routine MRI is also disappointing. It may infrequently show signal change on T2 weighted images or show enhancement following MR contrast agents but these are inconsistent findings. The benefit of CT and MRI in this setting is in excluding other causes of stroke-like symptoms.

Single photon emission computed tomography (SPECT) can demonstrate both fixed and reversible regional blood flow abnormalities in the brain. Provocative "stress" SPECT studies have been helpful in evaluating patients considered for endarterectomy with borderline stenoses. Its shortcoming is its relatively poor spatial resolution and cost.

Similarly, positron emission tomography (PET) can demonstrate regional blood flow abnormalities and metabolic disturbances, e. g., glucose metabolism. Although it has proven useful as a research tool, its very high costs, on-site cyclotron requirement to provide radionuclides, and other sophisticated requirements have limited its use primarily to academic centers.

Perfusion and diffusion MRI are rapidly developing techniques that show much promise in the evaluation of reversible ischemia. They utilize newer contrast agents and techniques that can image very early abnormalities in regional blood flow and intracellular edema that occurs with ischemia.

As newer therapies develop, their proper utilization will depend on the early identification of reversible areas of ischemia.

### STROKE

Persistent mismatch of regional metabolic needs (glucose, O<sub>2</sub>) relative to perfusion leads to irreversible injury. This leads to transmembrane dysfunction allowing accumulation of

intracellular water (cytotoxic edema).<sup>12</sup> Cytotoxic and vasogenic (extracellular) edema lead to mass effect. Blood brain barrier (BBB) disruption which leads to vasogenic edema is also a major influence of contrast enhancement patterns.<sup>13</sup> This simplistic pathophysiologic outline helps to understand the major CT and MRI findings in irreversible ischemia.

## CT

The sensitivity of CT in cerebral infarction is secondary to decreased x-ray attenuation due to increased tissue water.<sup>14</sup> The earliest density change can be seen at six hours but more typically occurs at 24 to 48 hours post ictus. Mass effect peaks at two to four days, although this is variable depending on infarct size. Peak enhancement occurs at seven to 10 days but may last several weeks. Arterial enhancement may occasionally be seen due to slow flow early in the post ictal period.

## MRI

The earliest change may occur immediately post ictus. These signs are secondary to slow flow or vascular occlusion. This causes the loss of normal vascular flow void and may lead to arterial enhancement (Figure 2).

Two to six hours post ictus early cytotoxic edema leads to subtle mass effect changes on T1 images. At approximately eight hours signal changes occur on T2 weighted images secondary to BBB breakdown. T1 signal changes occur at approximately 24 hours. Mass effect is similar to CT (Figure 3).<sup>15</sup>

At approximately seven days, progressive parenchymal enhancement occurs and may last for several months. Its onset correlates with the maturation of neovascularization in the repair process (Figure 4).

Early parenchymal enhancement correlates with a more favorable prognosis. It is felt to be secondary to BBB dysfunction, with an intact vascular supply. T2 signal changes are less apparent in this setting.

Chronic infarcts are characterized by atrophy, encephalomalacia, and the lack of mass effect and enhancement.

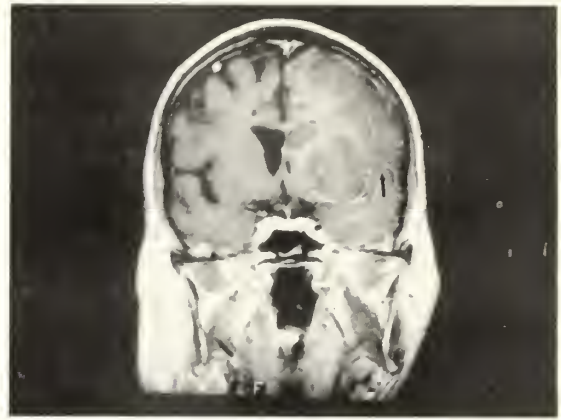


Figure 2. Acute left MCA infarct. T1 weighted coronal MRI of the brain following gadolinium enhancement. There is moderate decrease in signal intensity of cortical gyri secondary to cytotoxic edema causing sulci effacement. Arterial enhancement especially in the sylvian fissure (arrow) is seen secondary to slow flow due to peripheral emboli. Normally, high flow arteries do not enhance.



Figure 3. Acute left hemispheric infarct. T2 weighted axial MRI brain image shows hyperintense signal in left MCA distribution. A hemorrhagic component (dark area, arrow) is seen. It appears dark due to methemoglobin.

## CT vs MRI

MR is generally considered more sensitive than CT in the first 24 hours in the setting of acute stroke. MRI's multiplanar capacity, lack of bony artifact, and more frequent vascular changes makes it the procedure of choice in this setting.

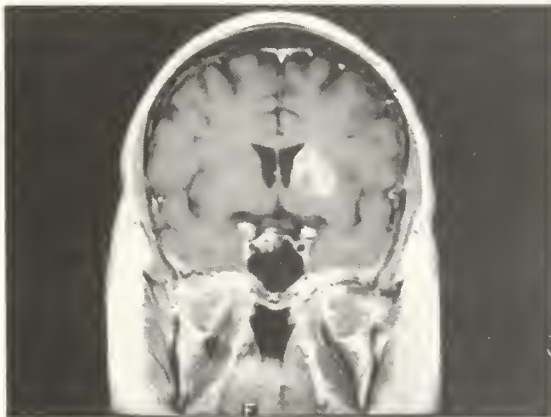
CT strengths are its relative lower cost, availability and easier patient monitoring.

Previously, CT was felt to be more sensitive to MR detection of hemorrhage. This may no longer be the case with newer tech-



**TABLE 1**  
**FINDINGS IN ACUTE STROKE**

<b>CT</b>	Density change 24-48 hours	Mass effect peaks 2-4 days	Contrast enhancement peaks 7-10 days
<b>MR</b>	Absent flow void Vascular enhancement immediate	T1, T2 signal change 6-24 hours	Parenchymal enhancement 5-7 days. Early parenchymal enhancement may correlate with a more favorable prognosis.



**Figure 4.** Subacute Infarct. Marked enhancement of the caudate and putamen nuclei following gadolinium.

niques (Table 1).<sup>16</sup>

In summary, the above and future advances in medical imaging are critical in effecting newer therapies which will decrease the morbidity and mortality of cerebral ischemia. □

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# COLOR FLOW DUPLEX SCANNING IN EVALUATION OF CAROTID ARTERY ATHEROSCLEROTIC OCCLUSIVE DISEASE

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PEGGY BROWN

The ability of the vascular lab to recognize and diagnose patients with significant carotid artery atherosclerotic occlusive disease is essential given that 50 percent of all ischemic strokes are the result of disease located in the extracranial carotid bifurcation. In order to identify patients at risk of stroke and prevent the incidence of carotid related strokes, the underlying carotid pathology must be identified in a safe, cost effective and noninvasive manner. Color flow duplex scanning meets these goals by providing both anatomic and physiologic information of the extracranial carotid vessels.

## WHY IS DUPLEX SCANNING NECESSARY? THE SCOPE OF THE ISSUE

Why perform elective evaluation of the extracranial carotid arteries? If the natural history of strokes is to be altered, it is necessary to understand the impact of this disease in the U. S. and have a method of identifying patients at risk for cerebrovascular accidents. Duplex scanning provides this method.

In the U. S. there are approximately 500,000 new strokes per year resulting in 200,000 deaths. The prevention and treatment of strokes presents a formidable challenge to the practicing clinician given that 50 to 90 percent

of patients have no warning symptoms prior to their neurologic event. Death occurs in 15 to 33 percent of all initial cases.<sup>1-3</sup>

Asymptomatic carotid stenosis patients (greater than 75 percent) have a combined TIA and stroke rate of 10.5 percent per year<sup>4</sup> which is similar to the stroke rate in symptomatic patients. Perhaps the most important study to date concerning the treatment of symptomatic carotid artery disease was the NASCET study (North American Symptomatic Carotid Endarterectomy Trial Collaborators) published in *The New England Journal of Medicine* in 1991.<sup>5</sup> In this study, the medical treatment for symptomatic patients (greater than 70 percent stenosis) resulted in a 26 percent stroke rate over 24 months compared to a nine percent stroke rate in patients treated with carotid endarterectomy. Carotid endarterectomy resulted in a 17 percent absolute risk reduction and a 65 percent relative risk reduction for fatal or major ipsilateral stroke. The efficacy of surgery for symptomatic patients with high grade stenosis was so strong that the clinical trial was terminated and a clinical alert sent to physicians advising them of the beneficial effect of surgery.

Duplex scanning has played a significant role at our institution in the treatment of these patients as a modality which accurately assesses the extracranial carotid vessels in terms of the presence and extent of atherosclerotic disease such that appropriate therapy can

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be instituted. Between 1971 and 1991, 1,100 carotid endarterectomies were performed by surgeons of the Carolina Vascular Institute at Self Memorial Hospital in Greenwood, South Carolina, with a 0.4 percent peri-operative mortality and a 3.4 percent stroke rate. Eighty-five percent of these patients were symptomatic. Of the asymptomatic patients, 84 percent had greater than 90 percent stenosis. Duplex scanning has been an invaluable tool to diagnose carotid disease and provides long-term followup for the post-operative patient.

### **WHAT IS A DUPLEX SCAN?**

A color flow duplex scan combines in a single device both real time, B mode ultrasound and color flow Doppler, allowing a simultaneous examination of the anatomic and physiologic features of the extracranial carotid system. Real time B mode ultrasound provides the anatomic detail by visualizing the common carotid artery, internal carotid artery, external carotid artery, vertebral and subclavian vessels. Physiologic and hemodynamic information is obtained by spectral analysis of the Doppler flow signal. By utilizing the Doppler shift principle, both velocity and direction of blood flow can be determined and an estimation of the degree of stenosis can be obtained.

### **WHO PERFORMS DUPLEX SCANNING?**

Experienced, well-trained personnel are essential for accurate and thorough noninvasive testing of the extracranial carotid vessels. Each accredited vascular lab requires that a registered vascular technologist (RVT) perform such testing which would include duplex scanning. A registered vascular technologist has successfully passed the registry exam and has achieved the professional standard of practice in the area of vascular technology. The R. V. T. credential indicates that the technologist has a good understanding of the physical principles of vascular instrumentation and their applications to the diagnosis of vascular disease. At the Carolina Vascular Lab, noninvasive duplex scanning of the carotid vessels has proven to be an accurate

diagnostic tool with a specificity of 90 percent and a sensitivity of 95 percent.

A process was developed in 1990 to provide a mechanism for voluntary accreditation of vascular laboratories. This organization, sponsored by 11 medical organizations with an interest in vascular disease, is called the Inter-societal Commission for the Accreditation of Vascular Laboratories (ICAVL). The characteristics of vascular labs evaluated include physical facility, organizational activity, structure of the lab, credentials of the medical director and technical staff, actual documentation of study performed, including diagnostic criteria used and the quality assurance utilization program. At the present time, there are seven vascular labs in South Carolina which have achieved accreditation, with the Carolina Vascular Lab being one of the first 24 accredited labs nationwide.

### **THE DEVELOPMENT OF THE DUPLEX SCAN**

To better understand duplex scanning as it presently exists, it is helpful to examine the development of this modality. With the arrival of carotid surgery and arteriography in the 1950s, it became necessary to provide a means of noninvasively evaluating the extracranial carotid arteries. Arteriography was not suitable for long-term followup and did not provide accurate detail of the vessel wall.

The first effort to noninvasively evaluate the carotid arteries involved continuous wave Doppler ultrasound which estimated blood flow velocities based on the Doppler shift principle.<sup>6-8</sup> This method was both subjective and nonreproducible as the operator could only recognize normal versus abnormal audible Doppler shift frequencies. The direction of blood flow was unknown with the continuous wave Doppler system and it was difficult to accurately localize the underlying vessel being investigated.

Pulsed Doppler evolved in order to address these problems.<sup>9</sup> Pulsing the emitted ultrasound frequency allowed more accurate iden-



tification and localization of the insonated vessel. By incorporating spectral analysis with pulsed Doppler,<sup>10-11</sup> the Doppler flow signals received could be analyzed and visually recorded as Doppler wave forms. It became evident that specific changes in these wave forms were associated with varying degrees of vessel stenosis and that each extracranial carotid artery had its own specific wave form characteristics. Thus, Doppler spectral analysis could not only help identify carotid vessels, but could also estimate a percent stenosis based on changes in Doppler frequencies.

Yet, the problem of visualizing abnormalities of the carotid vessel walls remained. Doppler spectral analysis could identify atheromatous plaques only if they were large enough to produce alterations of blood flow. Doppler analysis could not provide anatomic detail of the vessels in question. Small, rough, irregular intimal plaques having the potential for cerebral embolization went unrecognized. Real time B mode ultrasound allowed a logical means of visualizing the surface characteristics of vessels in both longitudinal and cross sectional views. A drawback of ultrasound was that it proved unreliable in determining vessel patency given that fresh thrombus and flowing blood have similar acoustical properties. Therefore, total occlusions of the internal carotid artery could go unrecognized by utilizing ultrasound alone.

Combining real time B mode ultrasound with spectral analysis of Doppler frequency shifts (Duplex scanning) addressed the problems by providing in a single device both anatomic and physiologic information about the extracranial carotid arteries in a noninvasive manner.

Recently conventional duplex scanning has been combined with real time Doppler flow mapping to simultaneously evaluate the anatomic and physiologic features of the carotid arteries (color flow duplex).<sup>12-14</sup> The flow map is color coded to indicate the direction of flow and flow velocity.

Color flow duplex scanning facilitates the identification of the carotid arteries and its

branches, as well as the vertebral arteries. Coils, kinks and total occlusions are more easily and rapidly identified and flow can be simultaneously evaluated in multiple vessels.

## CONCLUSION

The extracranial carotid artery can be accurately investigated in a noninvasive manner by utilizing color flow duplex scanning. The presence and significance of carotid artery atherosclerotic occlusive disease can be visualized and hemodynamic information obtained. This test is utilized as a screening tool for patients at risk of carotid artery atherosclerotic occlusive disease and is helpful in determining if the carotid bifurcation is the cause of strokes in patients suffering from neurologic events. □

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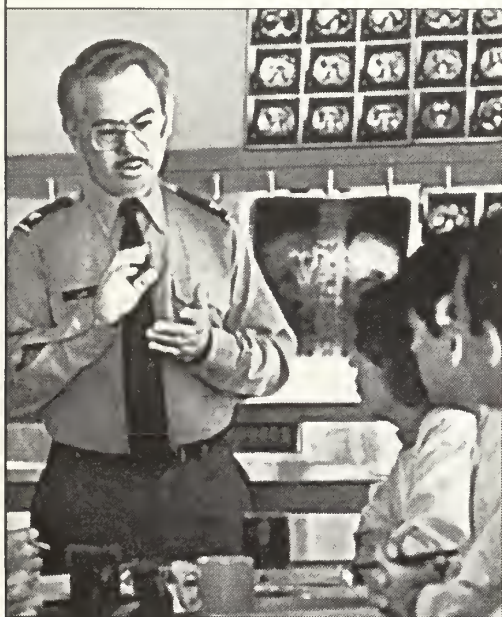
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# SCMA NEWSLETTER

A PUBLICATION OF THE SOUTH CAROLINA MEDICAL ASSOCIATION  
Joy Drennen, Editor  
798-6207, in Columbia  
Contributions welcomed  
1-800-327-1021, outside Columbia

July 1994

## MEDICARE UPDATE

By now you should have received the July, 1994 *Medicare Advisory*. Included in this *Advisory* is an update on Certified Nurse Midwife (CNM) Services, Hospice Part B guidelines, a clarification of prostate needle biopsy, and much more. Please read this *Advisory* carefully.

**Immunizations and Vaccinations:** According to Medicare guidelines, immunizations (with the exception of those for pneumococcal pneumonia, hepatitis B, and influenza) are non-covered services. Other vaccinations may be covered only when they are directly related to the treatment of an injury or direct exposure to a disease or virus. Preventive immunization is not covered. If a claim is submitted with an immunization code, there must be an indication that the patient has either suffered an injury which necessitates the injection or has been exposed to a disease or virus which requires the vaccination. CPT codes 90700-90749 are considered preventive medicine immunizations and are noncovered. You should bill with a "J" code when reporting an immunization that was medically necessary.

**Beneficiary Entitlement through Automated Response Unit (ARU):** You can now receive Medicare Part B Beneficiary Entitlement information through ARU. To receive information through the ARU, select Option 4 on the Main ARU Menu. The ARU will prompt you to enter the patient's Medicare number. After you've entered this information, the system will tell you if the beneficiary has Part B coverage and when that coverage went into effect.

**Provider Service Center Hours:** The Medicare Part B Provider Service Center is expanding its hours of operation. Effective July 1, 1994, lines will no longer close between 12:00 PM and 1:00 PM. New Service Center hours are: Monday through Thursday, 9:00 AM-4:00 PM and Friday, 9:00 AM-3:00 PM.

**Chemosurgery:** In the April, 1994 *Medicare Advisory*, CPT code 17305 (Chemosurgery [Mohs' micrographic technique]) was included in the list of codes which cannot be billed with modifier -58 (Staged or Related Procedure or Service by the Same Physician during the Post-

operative Period) during the global period. Medicare has determined that this code has a global period of "0" after-care days and each stage of the procedure has a different code (all of which have "0" global days). Therefore, CPT code 17304 is not subjected to the staged procedure exclusion and can be billed with modifier -58.

**Cystourethroscopy:** The descriptions of CPT codes 52234 through 52240 include the word "tumor(s)." HCFA interpreted these descriptions to mean that regardless of the number of tumors, only one unit of a single code may be billed. The correct billing and payment instructions for CPT codes 52234 through 52240 are:

- only one unit may be billed for any of these codes,
- only one of the three may be billed, and
- the billed code should reflect the size of the largest tumor removed.

No combination of the three codes may be billed on the same date of service.

**Rebundling Codes:** Medicare recognizes that the "rebundling" process, although correct for the majority of procedures performed, has resulted in some incorrect denials of services where the column II procedure was performed on a different anatomical site from the column I code. Medicare has contacted HCFA for assistance on how to correctly process these types of claims. In the interim, Medicare has developed the following solution:

When rendering services on different anatomical sites when the procedures are "bundled" together as noted in the December, 1993 *Medicare Advisory*, you should report each major procedure rendered at each anatomical site. If the procedure is a column II code, report the code with modifier -22 (Unusual Circumstances). You must file these claims hardcopy and attach documentation explaining the situation. Your claims will be reviewed to determine if additional benefits can be paid.

(Continued on page 2)

**CLIA Update:** Medicare will deny claims filed by Independent Clinical Laboratories (Specialty Code 69) who have not registered under the Clinical Laboratory Improvement Amendment (CLIA). Effective May 23, 1994, Medicare will also deny clinical laboratory claims filed by other non-physician specialties. See page 6 of the July, 1994 *Medicare Advisory* for a complete list of providers.

**Dialysis Monthly Codes:** Procedure codes 90918-90921 are for "end stage renal disease related services per full month." The reimbursement for these codes includes payment for an entire month. When billing for monthly dialysis services, you should report "1" in the Units field (item 24g) to indicate one full month of service. When filing a monthly service code, you should file only the first day of each month as the TO and FROM date of service. ☐

## MEDICAID UPDATE

**Medicaid Recipient Cards:** A problem has been identified with the transfer of ambulatory visit counts to the DSS computers for the printing of the recipient Medicaid cards. The column on the Medicaid recipient card that is headed "Est Visits Rem" (Estimated Visits Remaining) is an estimate of the visits the recipient has left at the time the card is printed. Some of the cards for April, May and June 1994 are not reflecting an accurate amount of ambulatory visits remaining. The problem is being

researched and corrective action is being made for the printing of future Medicaid cards. Please notify your staff of the problem and refer to your Medicaid Provider Manual if you receive error code 977 (ambulatory visits exceeded).

**Family Planning Prescriptions:** Any prescription classified as family planning will not count against the three prescription limit for Medicaid recipients. ☐

## SCIMER SAVINGS AND RETIREMENT PROGRAM

The SCMA, through its foundation, the South Carolina Institute for Medical Education and Research (SCIMER) has implemented an innovative savings and retirement program for physicians statewide. The Section 170 program was under review for over a year before being approved by SCIMER board. The Section 170 program derives its name from the fact that it is approved under Section 170 of the Internal Revenue code. It is a tax deductible gift annuity which allows the physician to make deposits in an account with SCIMER. SCIMER legally commits itself to pay a retirement annuity for two people for as long as either is alive. Also, the physicians' income stream is guaranteed by an A+ superior rated insurance company and reinsured by another A+ superior rated reinsurance company.

The vehicle used to provide income to the physician is a gift annuity which pays a fixed percentage rate. The physician gets an enhanced, guaranteed rate of return with safety and a tax deduction each year for the amount he deposits in his annuity. There are no limits on the amount the physician can contribute each year. The physician may deduct up to 50 percent of his or her adjusted gross income in any one year and carry forward any excess for five years. Income may be started any time without penalty, unlike a qualified plan. There are never any fees or charges with Section 170 and the physician has the flexibility to increase contributions or make lump sum year-end deposits. The program also takes contributions of stock or land to fund the program.

The physician creates an endowment in his name with SCIMER by giving the foundation permission to insure him or her. Somewhere in the future, at the demise of the physician, the foundation gets an endowment in the physicians' name. The foundation gets no current benefit from the physicians' contribution. Ultimately, all funds from the Section 170 program are used exclusively for programs in medical education and research for the benefit of physicians in the state of South Carolina.

*For additional information regarding the SCIMER Section 170 plan, call SCMA Headquarters and leave a message for Richard Howard or Brian Bock.* ☐



## PHYSICIANS CARE NETWORK UPDATE

Physicians Care Network has named Barbara Whittaker as Senior Vice President, and George H. O'Laughlin, of Summerville, has joined the Physicians Care Network staff as Director of Marketing. George has 10 years experience in health insurance sales specializing in managed care products.

To date, 14 hospitals have contracted with Physicians Care Network, and negotiations are pending with an additional 17 hospitals. The number of providers continues to increase with approximately 2,450 physicians enrolled.

Physicians Care Network staff is compiling a contact list of SCMA members who have an association with key persons in the business industry throughout the state. Information is forthcoming concerning how you can assist staff in its marketing efforts of contracting companies into the network.

## PHYSICIANS URGED TO CONTACT OSHA ON ENVIRONMENTAL SMOKE

The AMA, responding to tobacco industry efforts to generate "spontaneous" letters to OSHA protesting its proposed rules on exposure to environmental tobacco smoke, is urging physicians and medical organizations to contact OSHA as well.

The tobacco industry reportedly is generating up to 1,000 letters a day, and a strong response from the health care community is needed. Letters should come from state and local medical societies, specialty societies, and particularly from physicians who can relate individual case studies about the harmful effects of exposure to passive smoke.

*Send letters (with three copies) within 60 days to: OSHA Docket #H-122, Room N-2625, 200 Constitution Ave., NW, Washington, DC 20210.*

## DHEC CONTROLLED SUBSTANCES UPDATE

**Rescheduling of Talwin (Pentazocine):** Recent enactment of House Bill H.4872 rescheduled the oral dosage forms of Talwin (Pentazocine) as Schedule IV controlled substances in South Carolina; thus, Talacen, Talwin Compound and Talwin Nx may be dispensed pursuant to either written or oral prescriptions, and if indicated, such prescriptions may be refilled up to five times, or for a period of six months. Talwin injection remains in Schedule II and may only be dispensed pursuant to a written prescription or upon oral authorization in an emergency situation.

**Expiration Dates for South Carolina Controlled Substances Registration Certificates:** Recent enactment of House Bill H.4873 provides for changes in the expiration dates of South Carolina Controlled

Substances Registration Certificates. Physician registrations with certificates issued prior to July 1, 1994, will expire on October 1, 1994, instead of August 17, 1994, the date indicated on the current certificate. These registrants will renew and pay the annual renewal fee prior to October 1, 1994, not prior to August 17th, as indicated on the application which they will receive in July, and these registrations will expire on October 1, 1995 and October 1 of each year thereafter. Physician registrations with certificates issued on or after July 1, 1994 will expire on October 1 of each year thereafter.

*If you have any questions, please contact the Bureau Registration Section at (803) 935-7815 or by FAX at (803) 935-7820.* □



## **TOLL-FREE HOT LINE TO HELP COORDINATE DONOR INFORMATION**

The state's three primary organ and tissue procurement agencies have teamed up to establish a toll-free phone number for hospitals to use in referring potential organ, tissue or eye donors.

Beginning this month, hospital personnel may dial 1-800-269-9777, and they will be connected immediately with the SC Organ Procurement Agency, the American Red Cross Southeastern Tissue Services and the SC Lions Eye Bank. Health care personnel no longer have to remember three or four phone numbers, but may simply dial the Donor Referral Network's answering service at 1-800-269-9777. The service will contact the appropriate agency, thus making it easier for medical professionals to notify the agencies of potential donors.

This toll-free number is for use only by hospitals or other donor referral personnel and not for use by the general public. ☐

## **MANAGEMENT EDUCATION PROGRAM (MEP)**

Winthrop University, in cooperation with the American College of Medical Practice Executives (ACMPE), will offer the Management Education Program for Health Care Professionals effective September 16, 1994 on the campus of Winthrop University in Rock Hill. The program is a 12-module course "modeled on the traditional MBA program, designed specifically for physicians and administrators working in healthcare organizations." Meetings are once per month on Friday afternoon and Saturday morning, and the program content provides intensive courses which touch on all aspects of healthcare management, finance, marketing, management information systems and legal issues now affecting healthcare.

*For application form and/or additional information, contact Dr. Wilhelmenia Rembert at (803) 323-2204, or James Hacker at (803) 323-2186.*

☐

## **PUBLICATIONS/VIDEOTAPES AVAILABLE**

The SCMA Risk Management Committee has produced a revision of the tape, "Patients are People too." Two versions are available, one for a hospital setting and one related to a clinic setting. The tapes are useful educational tools for your office staff.

*To view either of the tapes on loan, contact Pam Taylor at SCMA Headquarters.*

☐

## **LEGISLATIVE UPDATE**

SCMA staff is currently working on a "Legislative Update" to recap the bills passed by the SC General Assembly which affect physicians and their practices. At press time, the governor had not yet signed the majority of the bills, although it is anticipated he will in the very near future.

**Watch your mail in the next few weeks for a complete legislative update.**

# THE SURGERY OF EXTRACRANIAL VASCULAR OCCLUSIVE DISEASE

EDWARD C. MORRISON, M. D.\*

Stroke remains the third leading cause of death in the United States and has a current annual incidence of 195/100,000 population.<sup>1</sup> The long term sequelae of stroke are not without significance: Nearly two-thirds of stroke victims are permanently disabled to some degree and nearly one-third of the long term survivors of stroke will require prolonged inpatient rehabilitation.<sup>2</sup> Current estimates suggest that one-half of the strokes in the United States are related to carotid distribution circulation and these strokes are closely associated with atherosclerotic involvement of the carotid bifurcation. Obviously the economic impact of stroke in the United States is burdensome (estimated 28 billion dollars annually<sup>3</sup>) and major collaborative efforts between neurologists and vascular surgeons have sought to identify not only those patients at risk for stroke but also those patients in whom carotid endarterectomy may reduce stroke incidence.

## SURGERY OF THE CAROTID ARTERY

Atherosclerosis is the overwhelming etiology of carotid bifurcation disease and although arterial fibrodysplasia does affect the carotid arteries, this rare entity accounts for less than five percent of carotid disease. Classification of the clinical syndromes of carotid disease into pleasantly organized categories continues to cause consternation among clinicians. Such terms as: TIA, RIND, stuttering hemiplegia, etc., and the often used Marseilles' Classification developed prior to the advent of widespread usage of CT scanning and magnetic resonance imaging. Thus, carotid symptom complexes need redefinition! Additionally with the widespread usage of Duplex scan-

ning, the tissue characteristics of carotid plaques (soft, fibrous, ulcerated, heterogeneous, etc.) are discernible and these characteristics will be found to have significant influence in the natural history of plaque progression and clinical symptoms.

Recently, a Report of the Ad Hoc Committee to the Joint Council of the International Society for Cardiovascular Surgery recommended practice guidelines for carotid endarterectomy (CEA) for the full spectrum of symptomatic presentations: transient ischemic attacks, prior stroke with minimal residual deficit, asymptomatic carotid stenosis, global ischemic symptoms, acute stroke, and stroke in evolution.<sup>4</sup> This excellent review provides a logical classification of carotid disease and thoroughly examines natural history, clinical evaluation techniques, historical results of medical and surgical management, and provides guidelines for appropriateness of carotid endarterectomy. Obviously, the breadth of the Ad Hoc report is beyond the confines of this brief review but it is strongly recommended to the clinician with interests in vascular surgery!

## TECHNIQUES OF CAROTID ENDARTERECTOMY

The technique of carotid endarterectomy (CEA) has been described since the late 1950s<sup>5</sup> and surgeons have developed various nuances and principles in performance of the CEA. Anesthetic techniques range from straight local, to cervical block, to general endotracheal. Arterial pressure monitoring is a standard, however, monitoring of the electroencephalogram (EEG) remains controversial. Full 12 lead EEG monitoring can be very useful with appropriate intraoperative interpretive support, however, the commercially available

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6 lead monitors (Lifescan) can lead to false complacency because of technical shortcomings of limited cortical monitoring. The utilization of intraoperative shunting continues to rouse controversy; the usual selective criteria include clinical status of the patient, angiographic status of contralateral carotid and physiologic status: inadequate stump pressures or intraoperative changes in EEG. The proper technique of CEA necessitates an endarterectomy through the outer planes of the media and a tapering of the intima into the internal carotid artery without the disastrous threat of an intimal dissection. Closure of the artery as well remains controversial with proponents of primary closure or patch angioplasty (prosthetic vs. vein). Completion monitoring of CEA has evolved from no monitoring to continuous wave Doppler to operative angiography and most recently, intraoperative duplex scanning for flow or intimal abnormalities.

It is a forgone conclusion that until prospective randomized, blind studies are available, the superior technique of CEA will be fleeting. Until then, each surgeon must evolve a protocol with which he is comfortable and which will also yield results that fall within national norms. Additionally, any hospital in which CEA is performed must provide a competent surgeon with excellent anesthetic support, a seasoned operative team and updated postoperative facilities in order to abide by recommended national standards.<sup>6</sup>

### **SURGERY OF THE SUBCLAVIAN-VERTEBRAL ARTERIES**

Surgical reconstruction of the subclavian and vertebral arteries is performed less frequently than carotid reconstruction, however, posterior circulatory disturbances are an important facet of cerebrovascular disease. Although "the vertebral artery lies buried in a sea of generalities, prejudices, fear and ignorance,"<sup>7</sup> atherosclerotic narrowing of the vertebral and subclavian arteries can result in vertigo, ataxia, dizziness, syncope, bilateral paresthesias,

cerebellar infarction and arm ischemia. Within the last 20 years, a variety of surgical techniques have evolved to rectify aortic arch, subclavian and vertebral disease without the attendant morbidity of direct aortic arch reconstruction. These techniques include vertebral artery endarterectomy, carotid subclavian bypass, and subclavian and vertebral transpositions. Surgical appropriateness for these procedures includes those patients with disabling vertebral basilar insufficiency without significant carotid arterial occlusive disease, and patients with disabling subclavian steal syndrome.

### **SUMMARY**

The task of reducing the entire field of extracranial cerebrovascular disease to a review of this brevity may be Sisyphean. The surgical therapy of extracranial cerebrovascular disease has developed into an established mode with excellent support from major clinical trials. In regards to symptomatic carotid disease, the North American Symptomatic Carotid Endarterectomy Trial (NASCET), and the European Carotid Surgery Trial (ECST) have both strongly endorsed surgical therapy in patients with 70 to 99 percent stenosis. In the near future, results from two major asymptomatic trials, Asymptomatic Carotid Artery Stenosis Study (ACAS) and Asymptomatic Carotid Stenosis Veterans Administration Study (VA No. 167) should be available to illuminate the controversies of asymptomatic disease. Undoubtedly, new technologies such as duplex scanning and magnetic resonance angiography will have greater roles in the screening of our rapidly aging population for these diseases. Finally, vascular surgery, a major consumer of high technology in hospital resources, will be continually scrutinized by the regulating agencies in the future. It will behoove all interested parties to pursue in hospital registries for monitoring of outcome data in this evolving field. □



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# MEDICAL MANAGEMENT OF ISCHEMIC CEREBROVASCULAR DISEASE

JOHN W. PLYLER, M. D.\*

## INTRODUCTION

Stroke is our leading cause of disability in adults and the third main cause of death. With education, new therapies, and urgent intervention, it is estimated that up to 85 percent of all strokes can be prevented or their effects reduced. For South Carolina, in the heart of the high risk "stroke belt," the continued development of effective means of diagnosis and treatment of stroke is of crucial importance for public health, economy, and quality of life. Prevention of stroke and additional brain damage following stroke remains the most important aspect of stroke management. While there are no treatments with clearly reproducible positive results, there are treatment programs which have demonstrated positive trends in prevention and outcome; new areas of treatment and prevention are becoming available.

## PREVENTION AND MANAGEMENT

Modification of risk factors is the mainstay of stroke prevention treatment. Table 1 lists risk factors and their relative risk for stroke based on mega analysis of clinical data available.<sup>1</sup> Other risk factors include infectious diseases such as syphilis, TB, endocarditis; inflammatory disease such as lupus and anti-phospholipid syndrome; drugs such as cocaine and high dose estrogens; blood dyscrasias; and potential risks have been reported with elevated uric acid, climate, stress, high sodium and low potassium, and sedentary lifestyle. Management of risk factors is reviewed.

### A. Previous TIA or CVA

Individuals with previous events are at high risk for stroke: those with crescendo TIAs,

prolonged events (>1 hour in duration), or recent events (first week after a TIA).<sup>2</sup> Appropriate management is individualized on a case by case basis with understanding of the pathophysiology of the clinical subtype of vascular ischemia coupled with awareness of potential interacting or limiting variables such as age and concurrent illnesses. The main cause of death in these high risk patients is cardiac infarction or sudden death. Currently, evaluation and treatment for coronary artery disease must be on a case by case basis; no clear cardiac management has been established for this group and more extensive research is needed to determine proper treatment. As aspirin and ticlopidine have been shown to have value in prevention of infarction, these are commonly prescribed in these high risk groups.<sup>2</sup>

Carotid endarterectomy has been shown to significantly lower risk of ipsilateral stroke in patients with >70 percent stenosis of the internal carotid artery. The role for surgical intervention in moderate disease and asymptomatic cases has not been established; medical treatment remains the current main therapy in these groups.<sup>3</sup> A number of studies have shown usefulness of antiplatelet-aggregating drugs following endarterectomy and many prescribe these post-operatively. Anticoagulant and antithrombotic drugs and antiplatelet-aggregating drugs are the major medical therapies available for lowering risk in high risk TIA patients.

*1. Anticoagulant and antithrombotic drugs :* A common approach to the high risk TIA patient is a short course of treatment with intravenous heparin followed by maintenance medical therapy or endarterectomy. While the rationale is strong, supportive data is limited and a large trial is needed to determine the usefulness of

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**TABLE 1**  
**TREATMENT ALGORITHM**

**Controllable**

(4-6 X risk)	1. High Blood Pressure –Most important controllable risk factor –Affects one in three American adults –140/90 is normal for adults
(2-6 X risk)	2. Heart Disease –especially Atrial Fibrillation –causes blood clots to form
(2 X risk)	3. High Red Blood Cell Count –Thickens blood and makes clotting more likely
(2-4 X risk)	4. Cigarette Smoking –Increases blood pressure –Causes platelets to stick together –Shortens platelet survival –Decreases clotting time –Increases blood thickness –Woman on birth control pills and smoke
(22 X risk)	5. Alcohol 6. Overweight 7. Cholesterol –200 or below is normal 8. Physical Inactivity

**Uncontrollable**

	1. Age –risk doubles every decade after age 55
	2. Sex –25% greater risk in males
(3 X risk)	3. Diabetes
(2-4 X risk)	4. Race –stroke 100% greater in blacks
(10 X risk)	5. Previous Stroke or TIA
(5 X risk)	6. Heredity –5 X (recent study of twins)
(2-4 X risk)	7. Asymptomatic Carotid Bruit

interim heparin therapy. Additionally, low molecular weight heparins may have pharmacological and therapeutic advantages but intensive study of these agents is needed.<sup>2</sup> The use of long term heparin therapy is unsupported by clinical study, offers no recognized advantage over warfarin, and is not currently recommended. The most widely used anticoagulant is warfarin. While past reported major bleeding risks have ranged up to 22 percent, recent studies have shown lower risks of <2.5

percent with the use of low level anticoagulants, close monitoring of levels, and patient selection for appropriateness of therapy.

Long term anticoagulation is recommended in patients with prosthetic heart valves, severe rheumatic heart disease, and severe cardiomyopathies.<sup>6</sup> Recent studies have supported long-term anticoagulation on oral warfarin for patients with chronic atrial fibrillation.<sup>5</sup> It is recommended that warfarin be given for three weeks prior to elective cardioversion in



patients who have been in atrial fibrillation for over two days, and that anticoagulation therapy be maintained until four weeks of normal rhythm is established.<sup>5</sup>

Oral anticoagulation is second line treatment for atherosclerotic disease; adequate data is not presently available. Warfarin may be indicated for patients allergic or intolerant to aspirin or ticlopidine, patients with continued symptoms on antiplatelet therapy, or those who have slow flow states such as vertebrobasilar ischemia, high grade stenosis, or intracranial stenosis.<sup>7</sup>

**2. Antiplatelet-aggregating drugs:** The Antiplatelet Trialists Collaborative Group reviewed available clinical research data and found antiplatelet drugs are effective in risk reduction for stroke, MI, and vascular death. Aspirin, the most evaluated drug for stroke prophylaxis, is effective, inexpensive, and relatively safe; it has become the gold standard for medical management in ischemic cerebrovascular disease.<sup>2</sup> The optimal dose for aspirin has not been established; current recommended dosing is 325 mg, daily up to the 1300 mg, FDA approved daily dosing regimen. Ticlopidine is the first agent shown to be

superior to aspirin in stroke prevention. In the large clinical trial TASS, ticlopidine was demonstrated to have an improved risk reduction for stroke and death over aspirin with both initial and recurrent strokes in women as well as men. Ticlopidine selectively inhibits ADP-induced platelet-fibrinogen binding and subsequent platelet aggregation. It is generally well tolerated; the most frequent side effect is abdominal pain and diarrhea with the most serious potential complication being neutropenia.<sup>9</sup> CBC monitoring every two weeks for the first 90 days of treatment is recommended. Those who cannot tolerate ticlopidine or who are at risk for complications should be given aspirin unless otherwise contraindicated.

### B. Hypertension

Systolic and diastolic hypertension increase risk for all stroke types and pressures over 160/90 warrant close attention. The following Table 2 is a management program recommended by the NIH Joint National Committee on High Blood Pressure.

### C. Heart Disease

**TABLE 2**  
**TREATMENT ALGORITHM**

Lifestyle Modifications  
Weight reduction  
Moderation of alcohol intake  
Regular physical activity  
Reduction of sodium intake  
Smoking cessation  
Inadequate Response\*  
Continue Lifestyle Modifications  
Initial Pharmacological Selection:

Diuretics or Beta-blockers are preferred because a reduction in morbidity and mortality has been demonstrated.

ACE inhibitors, Calcium antagonists, Alpha-1-receptor blockers, and the Alpha-beta blocker have not been tested nor shown to reduce morbidity and mortality.

Inadequate Response\*

Increase Drug Dose      or      Substitute Another Drug      or      Add a Second Agent from a Different Class

\*Inadequate Response\*

Add a Second or Third Agent and/or Diuretic if not Already Prescribed

**TABLE 3**  
**RISK MANAGEMENT**

<b><u>Risk Factor</u></b>	<b><u>Treatment</u></b>
Hypertension	Weight reduction, NA+ and K+ intake regulation, Antihypertensive drugs
Heart Disease	Aspirin, Coumadin, Antiarrhythmic drugs
TIA and Previous Stroke	Aspirin, Ticlopidine
Carotid Bruits	Aspirin
Diabetes Mellitus	Glucose Control
Smoking	Cessation
High-Estrogen Therapy	Low-estrogen therapy
Inflammatory or Infectious Disease	Treat underlying disorder
Lipids	Diet, Lipid lowering drugs
Obesity	Weight control
Blood Disorder	Treat underlying disorder

Vascular disease, ischemic disease, heart failure, and arrhythmias are risk factors. Patients with severe cardiomyopathy, prosthetic valves, rheumatic heart disease may require acute and chronic anticoagulation therapy. Patients with infarction, akinetic segments and septal defects may need short-term anticoagulation, i.e. <3 months.<sup>2</sup> Arrhythmia patients are candidates for long-term anticoagulation with recent studies confirming benefits in chronic atrial fibrillation.

#### ***D. Bruits***

Carotid bruits indicate atherosclerotic disease. Surgery is the treatment of choice in symptomatic bruits with high grade stenosis of the internal carotid. The proper treatment of asymptomatic carotid bruits is currently under investigation in clinical trials. At present, aspirin is considered a reasonable prophylactic therapy for documented asymptomatic carotid atherosclerosis.<sup>2</sup>

The major risk factors and management have been reviewed. Table 3 outlines other treatment regimes. It is likely that risk factors interact in at least an additive fashion but more likely in a non-linear manner; overall risk is probably more complex and greater than the simple sum.<sup>2</sup>

### **ACUTE MANAGEMENT**

Recent studies and trials have shown that early treatment is a major factor in treating

vascular disease. A time of two hours or less is probably required to avoid deficit and some studies indicate the need for onset of treatment within ninety minutes of onset of symptoms. There is no clearly established medical treatment of stroke. Figure 1 demonstrates a suggested approach from a regimen outlined by Stephen Oppenheimer.<sup>11</sup> All patients need medical attention, DVT prophylaxis, treatment of complications, and measures for secondary prevention.

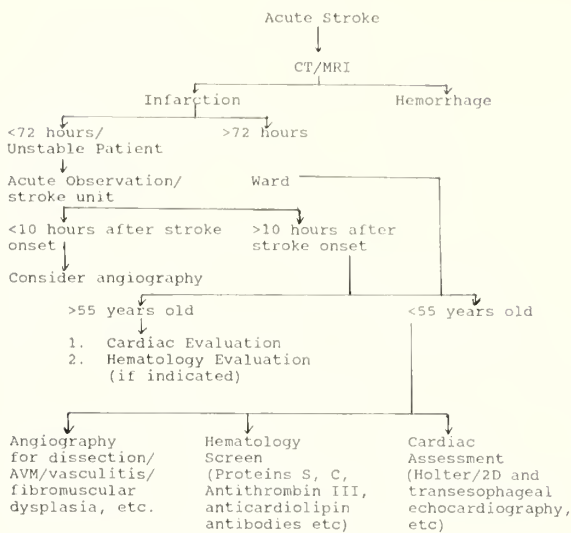
#### ***A. The ABCs***

Airway patency and circulation stability must be confirmed. Attention must be maintained for predisposing factors and possible cardiac injury. Because of likely lowered cerebral oxygenation and ventilatory function, supplemental oxygen of two to four liters per minute nasally is suggested.<sup>2</sup>

#### ***B. Blood Pressure***

Approximately 70 to 80 percent of stroke patients present with elevated blood pressure that reflects both hypertension as a risk factor and impairment of cerebral autoregulation. Table 4 outlines an approach to management of acute hypertension in stroke.<sup>7</sup> As a general rule, patients with pressures less than systolic 180 and diastolic 105 are not acutely treated in the first week of stroke unless there is the development of complications such as cardiac infarction, dissection or pulmonary edema. A

FIGURE 1



spontaneous decline in pressure may occur during the first week after stroke requiring close monitoring to avoid rapid drops which might cause decreased blood and oxygen to potentially viable brain tissue. Suggested guidelines indicate initial rate of decrease of pressure of 5-10 mm/hr for the first four hours and then 5-10 mm/4 hr until target pressure in the range of 160/95-100 or range of 180/105-110 in the known hypertensive patient. More aggressive intervention may be needed if there are associated complicating cardiac or medical illnesses. Long-term therapy goals and treatment programs may be considered after the first three days of acute therapy.<sup>4</sup> Hypotension may develop as a central effect and from arrhythmias and should be treated with antiarrhythmic and inotropes as clinically indicated.

### C. Glucose

Glucose level is elevated in 25 percent of stroke patients. While some are diabetics and in some the elevation is a stress response, a significant number are undiagnosed diabetics. Experimental and clinical studies have shown that chronic and transient hyperglycemia results in poorer outcome and intervention acutely is recommended with optimal glucose range being in the 60-90 mg/dl range; the administration of glucose in the acute phase should be avoided.<sup>2</sup>

TABLE 4

1. If systolic BP is 180-230 mmHg or diastolic BP is 105-120 mmHg, or both, emergency therapy should be deferred in the absence of documented intracerebral hemorrhage, acute myocardial infarction, or left ventricular failure.
2. If diastolic blood pressure (BP) is >140 mmHg on two readings 5-10 min. apart, start an infusion of sodium nitroprusside (0.5-10 mg/kg per minute).
3. If systolic BP is >230mmHg or diastolic BP is 121-140 mmHg, or both, on two readings 20 min. apart, give labetalol, 10 mg intravenously over 1-2 min. until a satisfactory BP reduction is achieved or until a cumulative dose of 300 mg has been administered by this minibolus technique. Labetalol is avoided in patients with asthma, cardiac failure, or severe cardiac conduction abnormalities. Alternative therapy may be considered with nifedipine, 10 mg orally or sublingually, but nifedipine should be used cautiously in stroke patients as an overly rapid decline in blood pressure is not common.

### D. Cardiac

Cardiac complications are the cause of up to 20 percent poststroke deaths. Cardiac monitoring in the first 24 hours is important and daily reviews of cardiac symptoms and initial signs are important. Routine laboratory injury profiles and daily EKGs are of low yield without other suggestive signs or symptoms.<sup>12</sup>

### E. Pulmonary Embolism and DVT

In stroke patients, pulmonary embolism may cause up to 25 percent of acute stroke deaths; therefore preventive measures are paramount and the following guidelines are suggested:<sup>2</sup>

1. Pneumatic compression boots for patients at bed rest.
2. SQ Heparin 5000 U every 12 hours if no contraindication.
3. Daily examination for evidence of DVT and respiratory symptoms.
4. Duplex ultrasound and/or VQ scanning in patients with positive or suggestive exams
- 5 Anticoagulation unless contraindicated if the diagnosis is established.

### F. Pneumonia

Aspiration is the primary cause of pneumonia in this patient group and may cause 15 to 25 per-



cent of stroke deaths. As preventive treatment, it is recommended that oral feeding be delayed until there is an intact cough on command and intact swallowing of sips of water. Signs suggestive of pneumonia warrant aggressive intervention and empiric therapy is recommended until a specific diagnosis is established.<sup>2</sup>

### G. Urinary Tract Infection

Almost half of stroke patients develop UTIs with most in association with use of indwelling catheters. Intermittent catheterization and condom catheterization are recommended if possible and systemic treatment reserved for treatment of systemic symptoms.<sup>13</sup>

### H. Skin

Decubitus ulcers are a frequent complication and potential source for infection. Preventive measures include maintaining dry skin, frequent turning, good nutritional support; high risk patients should be kept on fluid or air filled mattresses. If ulcers develop, wet to dry dressings should be applied every six hours and antibiotics and debridement may be needed if this regimen fails.<sup>2</sup>

### I. Cerebral Edema

Edema formation in cerebellar infarctions may be rapid and require surgical removal of the infarcted cerebellar hemisphere. Medical treatment includes osmotic diuresis with mannitol 25-50 mg. IV every three to five hours with supplementation if needed with furosemid 20-80 mg. IV every four to 12 hours. Fluid replacement should be aimed at maintaining osmolality at 300-320 mosm/L range and hypotonic or glucose fluids avoided. Mechanical ventilation may be needed for ventilatory support and acute short term effort at minimizing intracranial pressure. Edema in hemisphere infarcts usually becomes problematic on day two to five. Medical therapy is similar to that for edema in the cerebellar infarction except proven benefit remains to be clearly demonstrated and use of osmotic agents is usually reserved for large infarctions (based on individual case analysis). Steroid

therapy has no proven benefit in ischemic infarction as shown in a number of studies, some studies showing increased complications, and now is generally not recommended. Mechanical ventilation should be reserved for respiratory support.<sup>2</sup>

### J. Acute Medical Therapy

Heparin is widely used despite no clinically clearly established advantage. Currently accepted uses are for patients with strokes in evolution and cardioembolic stroke. Various regimens have been used with the following offered as a widely used program: an initial dose of 5000 to 10000 U followed by continuous infusion of 1000 u/hour; every 12 hours a PTT should be checked and rate adjustments made to keep a PTT level of 1.5 to 2.0 times control. Heparin is generally continued for 48 hours and replaced by oral anticoagulants if indicated.<sup>14</sup> Warfarin is not used initially as it takes several days to reach appropriate levels of anticoagulation. Long-term heparin therapy is unsupported and long-term warfarin use has been earlier discussed. Many new therapies are under investigation and offer promising treatment alternatives; a low-molecular weight heparinoid is a treatment available in S.C. under the NINDS multicenter TOAST study (contact Dr. Tim Carter or Dr. John Plyler).

**K. Secondary Prevention** In TIA, mild CVA, and even severe stroke patients, evaluation for

FIGURE 2

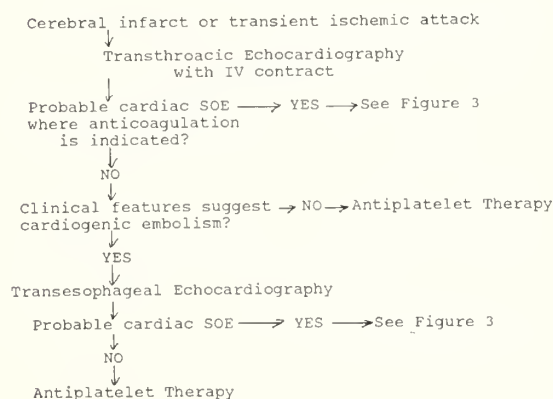
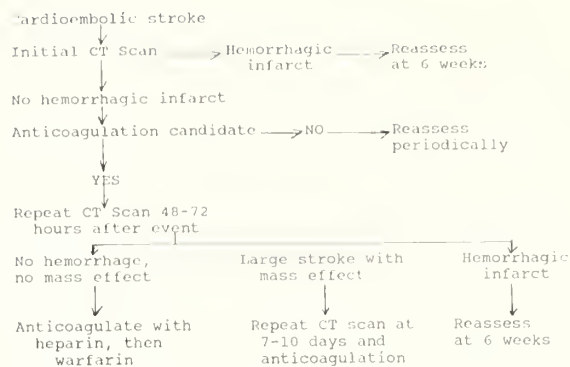


Figure 3



secondary prevention of recurrence is important. The use of ASA, ticlopidine, and endarterectomy have been discussed in earlier sections. Adequate data for secondary prevention of cardioembolic stroke is not yet available. Probable causes include arrhythmia, valvular abnormalities, and myocardial abnormalities which increase the likelihood of thrombus formation. Figures 2 and 3 provide a suggested treatment scheme outlined in *Current Therapy in Neurological Disease* and offer a reasonable approach to management of cardioembolic stroke.<sup>11</sup> Probable sources are generally treated with long-term anticoagulation; exceptions include recent MI (three to six months treatment), normal sinus rhythm after conversion (four to 12 weeks), and sick sinus syndrome (case basis). Patients with possible sources are generally treated with antiplatelet therapy.

## SUMMARY

Treatment of ischemic cerebrovascular disease is a complex area requiring multi-level assessment and intervention. The need for urgent acute and aggressive intervention is

clear. The armamentarium in stroke treatment is growing with new therapies under active investigation including thrombolytic agents, NMDA antagonists and amino acid blockers, and for radical scavengers. Very likely a multimodality medical therapy approach will evolve and the time has arrived for aggressive care of the stroke patient. □

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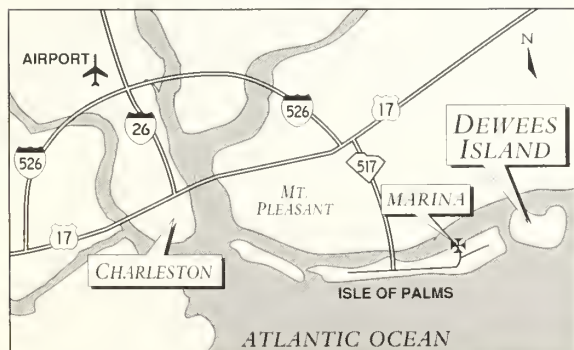
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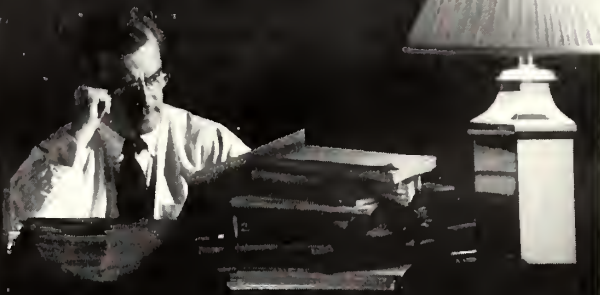
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**Contraindications:** Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

**Warning:** Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

**Adverse Reactions:** Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.<sup>1,2</sup> Also dizziness, headache, skin flushing reported when used orally.<sup>1,3</sup>

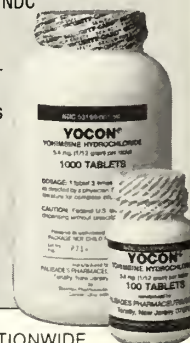
**Dosage and Administration:** Experimental dosage reported in treatment of erectile impotence.<sup>1,3,4</sup> 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to 1/2 tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.<sup>3</sup>

**How Supplied:** Oral tablets of Yocon<sup>®</sup> 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

#### References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical Letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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## THE MAGNITUDE OF THE PROBLEM

Arterial diseases are a leading cause of death in the United States and many other countries. Probably more important than mortality is the severe disability that disease inflicts on many people. For example, it is estimated that 400,000 to 500,000 first strokes occur in the United States annually. The social and economic impact of a stroke can be devastating to both the patients and their families. Other individuals are incapacitated by angina pectoris, leg claudication and ischemic foot disease.

Atherosclerosis is by far the most common cause of ischemic cerebrovascular disease. Other etiologies such as cranial arteritis and fibromuscular hyperplasia are also very important.

Researchers found that the most reliable indicator of stroke proneness resulting from atherosclerosis is evidence of ischemic heart disease manifested by electrocardiographic changes, angina pectoris, previous myocardial infarction, or congestive heart failure. Many reports emphasize the fact that every patient who has symptomatic cerebrovascular disease must be evaluated for disease of the heart, aorta or cervical arteries, as well as for abnormalities in the intracranial arteries themselves.

Some believe that the prevalence of atherosclerosis in the intracranial arteries varies enormously from one geographic region to another ("stroke belts"), as well as among ethnic groups within one area. Others disagree with these conclusions.

In men, atherosclerosis affecting the coronary arteries usually becomes symptomatic between the ages of 45 and 55 years, while similar changes in the cervical and cranial arteries start at 55-65 years of

age. Women, on the other hand, have their initial symptoms later in life, leading one to believe that either a hormonal relationship or a difference in exposure to environmental risk factors play a role.

There has been a remarkable decline in the incidence of stroke during the past 15-20 years. It is largely attributed to the early detection and advances in successful treatment of arterial diseases, particularly aggressive management of hypertension which not only is a very important factor linked to intracranial hemorrhage, but also accelerates atherosclerosis. The difference is also related to a decreased use of tobacco products, modification of diet and recognition of risk factors and prevention. Several studies have shown that the risk of ischemic stroke is two to four times higher in smokers than in nonsmokers, for both men and women. The risk of cerebral hemorrhage, either parenchymatous or subarachnoid, is particularly striking: smokers have a ten-fold greater risk of cerebral hemorrhage than nonsmokers. Similar increased risk of hemorrhagic stroke is associated with alcohol consumption. Another reported risk factor for hemorrhagic stroke is a low serum cholesterol level.

It is important for us, as physicians, to ascertain the presence of other known risk factors (i.e., use of oral contraceptives, presence of antiphospholipid antibodies, hypercoagulable states, atrial fibrillation, orthostatic hypotension, migraine headache, mitral valve prolapse, or substance abuse) and to apply preventive measures when possible. These changes are, unfortunately, slow and a problem of ischemic cerebrovascular disease will remain a very

important health issue during the entire careers of most physicians, even the youngest ones.

Although in decline, stroke still ranks third, after heart disease and cancer, as the underlying cause of death in the United States. Six of 10 victims survive. Rates of strokes for blacks are generally higher than those for whites, and rates for men tend to approach the maximum and those for women the minimum estimates. For every 100 survivors of stroke, 10 will be essentially normal, 40 will have mild residual disability interfering with motility, employment or daily living, 40 will be disabled severely as to require special care, and 10 will require institutional care. Across the country, figures show that stroke victims occupy 20 percent of all skilled nursing home beds. Stroke is not only costly because of its fatality, physical and mental impairments it causes, but also because of its economic burden. In our

nation, 25 percent of stroke survivors are below 65 years of age. According to National Survey of Stroke, the cost of stroke, considering direct and indirect expenditures and economic losses engendered, are close to nine billion dollars each year.

At the end, I would like to thank each contributing author to this issue: Timothy D. Carter, M. D., Jerome E. Kurent, M. D., Jim E. Fogartie, Jr., M. D., Michael C. Garovich, M. D., Edward C. Morrison, M. D., and John W. Plyler, M. D. As in the first issue, my special thanks go to the staff from the Editorial Office of the South Carolina Medical Association, as well as to Ms. Betty Newsom from the Waring Historical Library at Medical University of South Carolina.

Artur Pacult, M.D., F.A.C.S.  
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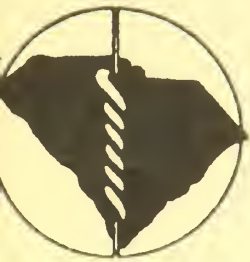
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# THE ANNUAL MEETING OF THE AMA CHICAGO, ILLINOIS – JUNE, 1994

## REPORT OF THE SCMA DELEGATION

WALTER J. ROBERTS, JR., M. D.\*

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The annual meeting of the American Medical Association was held in Chicago on June 12-16, 1994. In attendance from South Carolina were members of the South Carolina AMA Delegation, including SCMA President Dr. Marion Burton who is an alternate delegate by virtue of his presidency. Also present were President-elect Dr. Ned Nicholson and Secretary Dr. Bryan Walker. Dr. Charles Geer of Charleston and Dr. Roger Ray of Anderson also were present attending the Hospital Medical Staff Section of AMA and Dr. March Seabrook and Dr. Dina Grice, both of Columbia were present at the Young Physicians Section of the AMA Meeting.

Finally, there were a number of medical students from both of our medical schools in attendance at the AMA Medical Student Section meeting. South Carolina was well represented in all these meetings, and took an active part in the important deliberations.

Just prior to the AMA meeting, I was privileged to preside at the annual meeting of the Organization of State Medical Association Presidents (OSMAP), as the outgoing president. I am pleased to say that I enjoyed greatly the

year as president, and felt that the experience was personally rewarding, and rewarding for South Carolina through our exposure nationally.

Speaking of national exposure, our member of the AMA Board of Trustees, Dr. Randy Smoak, continues in increasing prominence. As many of you saw on national television, Randy represented AMA recently in Congressional hearings on the tobacco and smoking issue, and has been widely quoted as a spokesperson on behalf of the physicians of the country in this important debate.

We have also been pleased to hear recently of the appointments of two South Carolina physicians to national offices: Dr. Dina Grice to the Advisory Committee on Women in Medicine, and Dr. Ken DeHart of Horry County to the CPT Editorial Board. These appointments give added prestige to South Carolina nationally.

### HEALTH SYSTEMS REFORM

It should certainly come as no surprise that the major topic for discussion, and the major source for reports and resolutions involved the current debate on

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\*207 Spring Valley Road, Columbia, SC 29223

Health Systems Reform. As you know, the Clinton Administration's reform bill is, by everyone's admission, in deep trouble and unlikely to be accepted in anything like its original form by the American people. At the same time, several other health reform proposals, running a spectrum which ranges from "single payor" systems resembling the Canadian Health Plan to numerous modifications of the Administration's plan have been advanced by members of Congress. To this point, none appear to be acceptable, principally because of lack of "Voice and Choice" or because of the absence of any culpable plan for financing.

In this setting, the state of Illinois presented the AMA House of Delegates with several resolutions which amounted to an attack — called a "conservative attack" by *AM News* — on several portions of the Clinton plan; and indeed, one of the resolutions called for the AMA to express opposition to the plan in its "current form." Another Illinois resolution called for AMA rejection of the idea of an employer mandate for providing health care insurance; still another asks that freedom of choice of MD/DOs be part of any state or national health insurance proposal.

Though it is unerringly clear that most physicians philosophically support such ideas, it has been agreed, by consensus and not without much debate, that AMA has postured itself to maintain dialogue with the Administration, as well as with Congress in the development of all Health Systems Reform strategies, and that to this point, the ability to do so has served

us well. The House of Delegates voted to "stay the course," to keep avenues of dialogue open between the White House and Congress so that we, as physicians, may be at the table instead of outside looking in during these monumental decision-making times for Medicine. Though many will consider this an attitude of passivity, nothing could be further from the truth.

At present, the approach of the American Medical Association toward Health Systems Reform may be summarized as follows:

- Continued surveillance — and continued dialogue with — the President's effort to obtain a bill which can garner enough support in Congress to become law. After all, it must be remembered that our own Health Reform proposals, *Health Access America*, formed the basis, with several alterations, for the President's original effort.
- Continued endorsement of a plan for financing Health Systems Reform which includes employer mandates, individual mandates and medical IRAs or some form of tax-advantaged savings account for medical expenses.
- Continued opposition to any and all "Single Payor" systems. (Representative McDermott came to the AMA meeting, and presented his plan for such a system. His session was well-attended, if not-so-well received. To my mind, I have to admit that his statistic indicating that 30 percent of the health care dollar is being siphoned off by the insurance industry was impressive. This amounts to some 300 billion dollars a year, if the statistic is correct, a savings which would at least correct the

financial problems related to health care expenditures.)

- Rejection of a proposal for a pure “any willing provider” measure, recognizing this as a “two-edged sword.” Although the penetration of HMOs in this part of the country is not nearly so great as on the west coast, or in states such as Minnesota, many physicians are involved in plans where a “come one, come all” measure would prove untenable and unworkable, they feel. The current AMA policy encourages HMOs to make available to any interested and potential panel physician their requirements for admission to that panel, and equally important, the provisions under which the physician's panel membership could be terminated.
- Allow and encourage physician-owned and physician controlled managed care organizations, such as we have begun with the South Carolina Medical Association Physicians Care Network. The attitude of the AMA toward managed care has clearly softened, and it seems that, at the very least, managed care is something we will have to live with. The sentiment of the House of Delegates was clearly that if we have to live with managed care and if managed care is going to dominate the health care market of the future, physicians should be in charge.
- Continued support for fee-for-service medicine, despite this apparent acceptance of managed care. It was recommended that if employers or insurers provide “menus” of health care benefit plans, there should be at least two fee-for-service plans offered.

The House of Delegates also expressed opposition to the managed care premise that specialty consultation should be available to patients only upon recommendation of a “gatekeeper.”

## ETHICAL OPINIONS

Two significant items regarding ethical opinions came to the Reference Committee on Amendments to Constitution and Bylaws, a committee I was privileged to chair at the Annual Meeting. Georgia and Illinois presented resolutions asking the Council on Ethical and Judicial Affairs (CEJA) to reappraise its opinion that it is unethical for a physician to accept drug samples for personal use, or for the personal use of family members. CEJA will do so, but it is my belief that the opinion will not be changed.

Also, an opinion of CEJA regards the use of organs for transplantation and for other usage from anencephalic infants as “ethical.” Principal concern was over the possible development of a “slippery slope” where infants with conditions other than anencephaly might become such organ donors. This concern can be addressed by careful diagnoses and monitoring, CEJA and the House of Delegates agreed.

## RBRVS

Confusion, frustration and disillusionment have been openly expressed once more in the way Medicare has attempted to implement RBRVS. A resolution was presented asking AMA to study the feasibility of AMA devel-



oping its own resource value scale. The resolution was accepted, somewhat to my surprise. It seems to me that this would be a prodigious undertaking. We will see what the Board of Trustees recommends at the Interim meeting, and I will report to you.

## ERISA

The Employee Retirement Income Security Act (ERISA) was passed in 1974, and was devised by Congress to exempt self-funded employee benefit plans from state mandates and controls. Originally designed to protect employee pension plans, the law now is blocking many states in their health reform efforts, and giving employers decisive control over workers' health benefit plans. A Board of Trustees report to the House of Delegates outlines the many possible negatives in ERISA usage, citing the employers' ability to influence decisions regarding compensation in cases of injury. AMA has already been testifying in efforts to modify these potentially

undesirable features of ERISA, and to aid state efforts in health care reform which may be obviated by ERISA regulations.

Please let me once more direct you to *AM News*, June 27, 1994, for more "in-depth" coverage of these and other issues discussed at the Annual Meeting of the AMA. Let me also assure you that you have a voice in AMA, and your delegation stands ready and willing to express that voice. These are difficult times in medicine, and the course which physicians can set for themselves for the coming years, the uncertainty surrounding the arena of medical education and research, and most important, the well-being and benefit of our patients are integral parts of the AMA decision-making process.

Stay involved!

Walter J. Roberts, Jr., M. D.  
Chairman, SCMA AMA Delegation

# On the Cover:

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## ANTONIO SCARPA 1747-1832

The magnificent plate on this month's cover is from Antonio Scarpa's anatomic masterpiece, *Tabulae Neurologicae ad Illustrandum Historiam Anatomicam* published in 1794. The plates were all drawn by Scarpa himself and are lifesized.

Scarpa was born in northern Italy. His early education was thorough, and by the age of 15 he was an excellent Latinist and had passed the entrance exams for the University of Padua. It was there that he came under the tutelage of the famous anatomist, Morgagni. At the age of 20, with Morgagni's blessing, Scarpa was named Professor of Anatomy at the medical college at Modena. His first published work described several anatomical structures which bear his name, the best known being Scarpa's triangle of the thigh. His *Tabulae Neurologicae* included the first proper delineation of the nerves of the heart.

He wrote a beautifully illustrated book on ophthalmology; distinguished true from false aneurysms and introduced the concept of arteriosclerosis; wrote important treatises on hernia and eye disease; and made a shoe for club-foot which still serves as a model for orthopedists.

Scarpa achieved wealth and fame during his lifetime. He was a favorite of Napoleon I, who made him an imperial surgeon and awarded him the Cross of the Legion of Honor and the Order of the Iron Crown. He died in his 80th year.

Betty Newsom  
The Waring Historical Library

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2. Rutkow, Ira. *Surgery, An Illustrated History*, 1993.

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# Alliance Page

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Medicine invades our home life. It places us into the public eye whether we like it not. It challenges us to the responsibility of "giving back" to the community.

SCMAA is an organization that is dedicated to creating community awareness of today's health issues and as partners in medicine, helping to improve the quality of life for **ALL** Americans. Public relations begins at home and each Auxiliary/Alliance member represents and is a spokesperson for the medical profession as well as the Alliance.

Time constraints may make active participation difficult for some, such as "Career Spouses." However, you have the potential of reaching a vast number of people. No one individual needs to do everything in Alliance but contributing to at least one facet of the organization is vital and valuable.

So I encourage all spouses to become involved and join our effort on the county, state and National levels. You will benefit personally by knowing that you support programs that do make a **DIFFERENCE** to thousands of people-children and adults alike.

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## INDEX TO ADVERTISERS

Ann M. Bowen, CFP .....	340
Companion Technologies .....	305
CompuSystems .....	Cover 4
Dewees Island .....	337
Harry A. Hancock, J. D., LL. M. ....	Cover 3
Lexus of Charleston .....	Cover 3
Medical Protective Company .....	322
Medical Management Systems, Inc. ....	338
Palisades Pharmaceuticals .....	338
T. Rowe Price .....	Cover 2
Roche Laboratories .....	306
U. S. Air Force .....	313
U. S. Air Force Reserve .....	346
U. S. Army Reserve .....	321
U. S. Navy Reserve .....	329
Winchester Surgical Supply Company .....	346

# Gray Matter

*"Matters of Interest  
to South Carolina  
Physicians."*

Thornton & Thorne give the medical community something to think about this month.

## **AMA INCREASES DISABILITY PREMIUMS (AGAIN)**

### **THIRD INCREASE SINCE 1990 INCREASES TOTAL 26%**

The American Medical Association offers disability insurance to its members through an *association group contract*. Like all group contracts, this one allows the premiums to be changed. *Premiums have increased three times since 1990.*

Current premiums are 26% higher than those projected prior to the 1990 increase. As a premium increase applies to all policyholders, insureds who have had their disability insurance with AMA will be paying premiums that are substantially higher than those projected when they purchased their coverage.

Premiums for AMA disability insurance are *scheduled* to increase in 5 year brackets based on age. The 26% increases we're referring to are *non-*

*scheduled* and are in addition to those based on age.

The contractual provisions of an individual disability policy are superior to those in any group policy. This begs the question *"if an individual policy provides better terms, why do physicians purchase disability insurance through the AMA group plan?"*

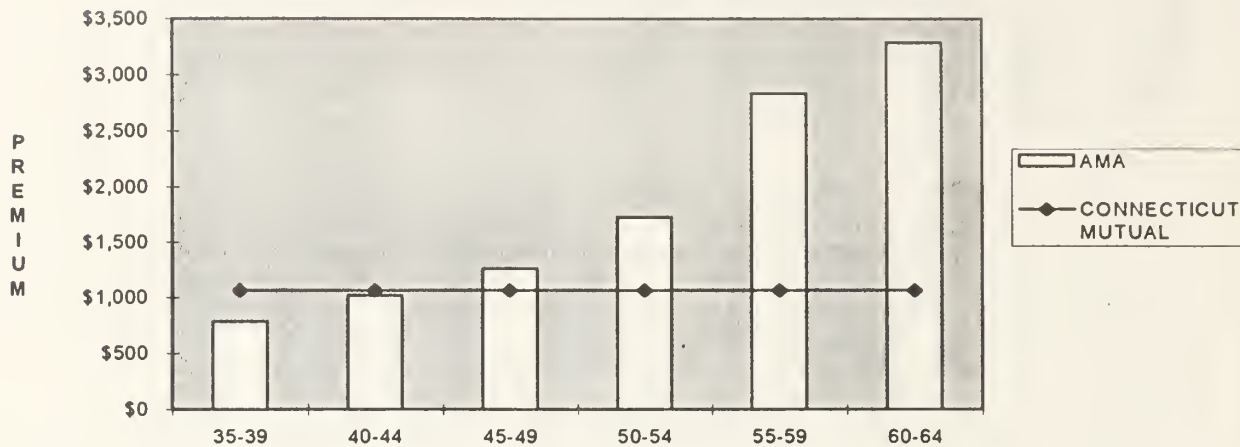
The apparent reason is that many physicians think the AMA policy will cost less. However, if outlay to age 65 is considered, an individual policy saves thousands of dollars over an AMA policy.

The graph on the following page shows that the outlay for a 35 year old SCMA member to carry an AMA policy to age 65 is substantially greater than the outlay to carry a policy issued by Connecticut Mutual. Other issue ages would experience similar results.



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### SUMMARY

The contractual provisions of an individual disability policy from Connecticut Mutual are superior to those of an AMA policy.

The outlay for a SCMA member to carry a Connecticut Mutual policy to 65 is *less* than the outlay for a policy from AMA.

Premiums for a Connecticut Mutual policy are guaranteed to age 65. Premiums for an AMA policy are guaranteed to September 30, 1994.

*If you have your disability insurance through the American Medical Association, you will receive better contractual terms and lower cost if you change to Connecticut Mutual.*

Views expressed herein are those of the authors and in no way represent SCMA.



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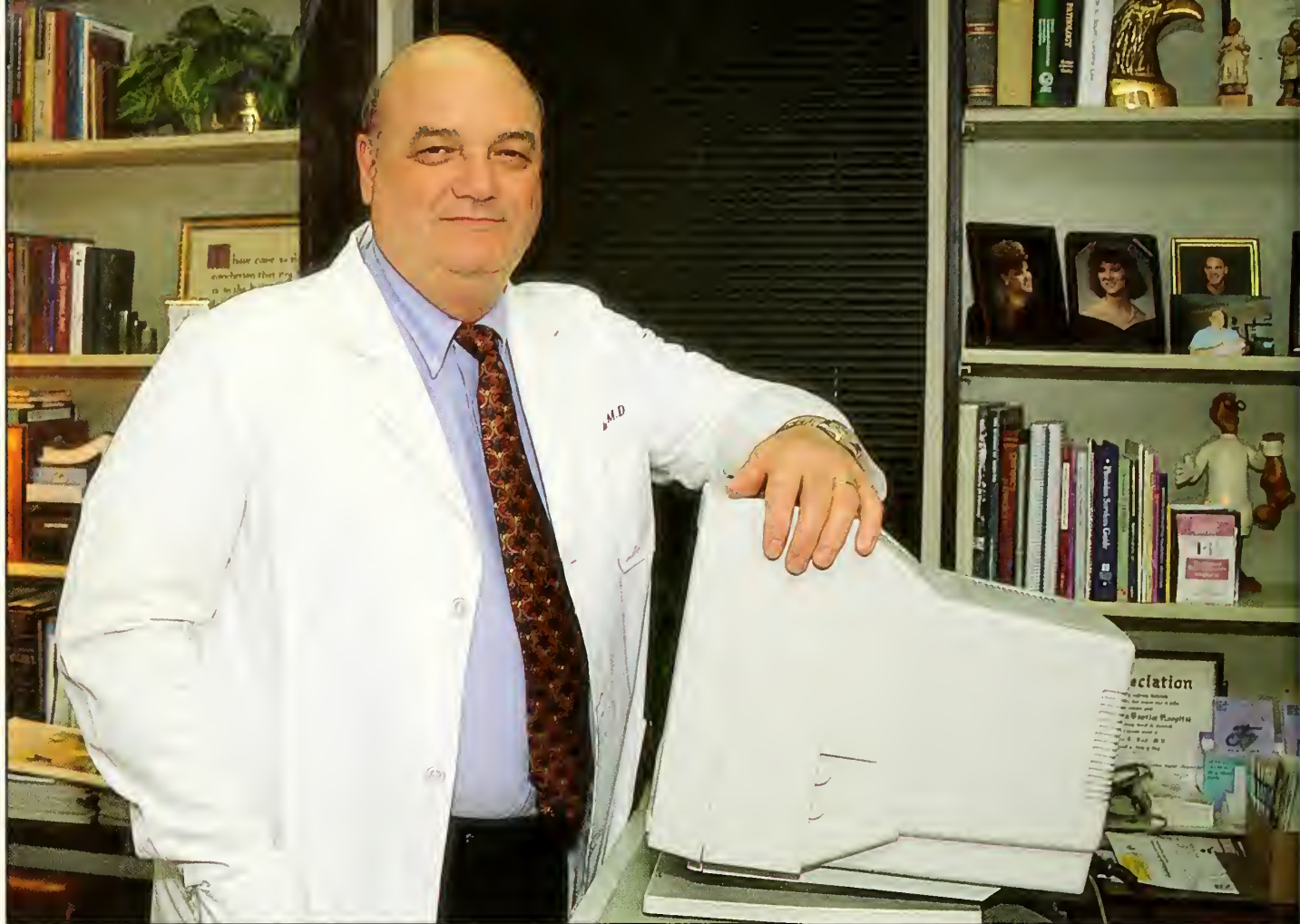
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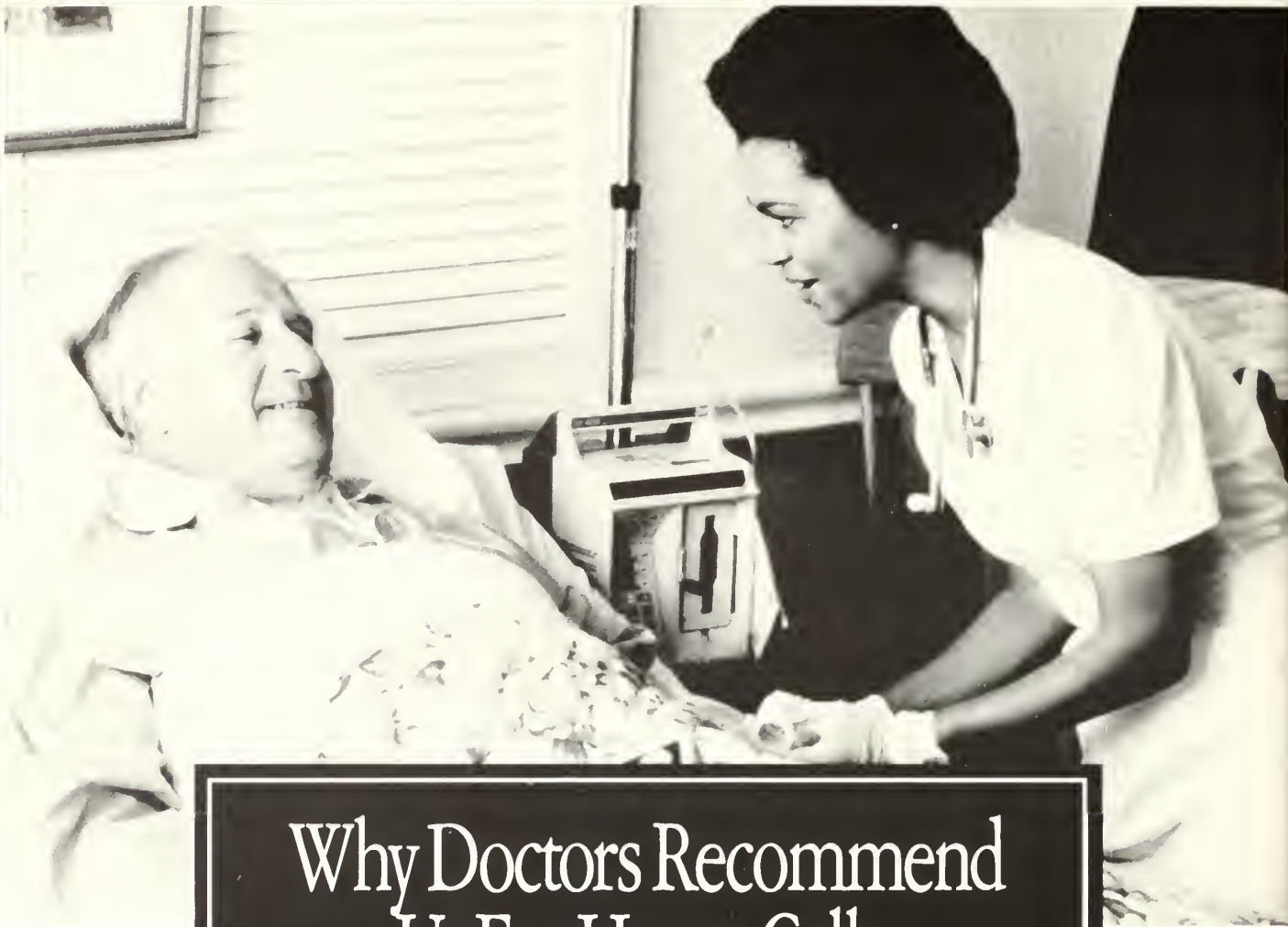
OF THE SOUTH CAROLINA MEDICAL ASSOCIATION

VOLUME 90  
NUMBER 8  
AUGUST 1994  
PAGES 351-392

**BICYCLE RELATED HEAD INJURIES  
IN VITRO FERTILIZATION AND EMBRYO TRANSFER  
ACUTE PSYCHIATRIC DISORDERS IN THE ELDERLY  
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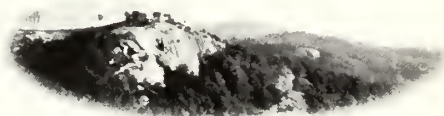
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# President's Page

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## LEGISLATIVE UPDATE

The final year of the 110th session of the General Assembly ended in havoc as lawmakers struggled to complete a budget amid controversy over the confederate flag, shortage of revenue and the future of the Barnwell Nuclear Storage Facility by the June 2 mandatory adjournment date. Between shots fired by House and Senate members of the Budget Conference Committee, several major bills affecting physicians and their patients were passed through delicate parliamentary maneuvering.

We received consistent support from our friends in the legislature. **Representative Billy Houck, M. D.**, introduced legislation which would allow patients the right to choose their physicians in any managed care plan. After intense lobbying by the South Carolina Chamber of Commerce and Blue Cross/Blue Shield of South Carolina opposing the bill, it was amended to the point of impotence and withdrawn by the author. The SCMA was astounded that business leaders would oppose freedom of choice and free market principles, lobbying strongly to create monopolies and anti-competitive market forces designed to diminish the physician-patient relationship.

The perpetual efforts by the South Carolina physical therapists to practice independently of the prescription of a physician was again defeated by the SCMA. The bill was sponsored by Representative David Waldrop (D-Newberry). In the House, opponents of the bill, led by **Representative Steve Lanford** (R-Spartanburg), killed the House measure along with an attempt by Representative Dell Baker (R-Greenville) on a possible compromise. Next, the Senate Medical Affairs Subcommittee, chaired by Senator James Bryan (D-Laurens), adopted the compromise which had been proposed by Representative Baker in the House. Voting with Senator Bryan was Senator Robert Ford (D-Charleston). **Senator Greg Gregory** (R-Lancaster) voted against the compromise and the bill. When it reached the Senate floor, **Senator Holly Cork** (R-Beaufort) objected to the bill and single-handedly killed the Senate version.

The feeding frenzy for “wannabe” doctors to grab a slice of the insurance pie continued at an alarming pace. A bill giving mandated benefits to doctoral clinical psychologists, opposed by the SCMA and the South Carolina Psychiatric Association, passed the legislature. In addition, a bill mandating insurance coverage for services offered by any licensed provider of mental health, such as marital and family therapists, social workers, etc., passed the House and was approved by the Senate Banking and Insurance Committee. The SCMA organized opposition to the bill. **Senator Greg Ryberg** (R-Aiken) objected to the bill, despite angry protests from supporters, and killed the legislation.

This past legislative session clearly demonstrates why every physician should be a member of SOCPAC. Please join today!

O. Marion Burton, MD  
*President*

## Contents

### Original Scientific Articles

- 355 Bicycle-Related Head Injuries in South Carolina Children: What Can We Do to Prevent Them?**

*Neal J. Thomas, M. D., Janice D. Key, M. D., Walton L. Ector, M. D.*

- 360 In Vitro Fertilization and Embryo Transfer (IVF-ET) Comes to the Upstate**

*William R. Booue, Ph. D., Jane E. Jolusou, M. F., Thomas M. Price, M. D., Betty K. Bowers, M. N., M. Dawn Morris, B. S. N., Martin M. Craue, Ph. D., William Bates, M. D.*

- 373 A New Approach for the Management of Acute Psychiatric Disorders in Elderly Demented Patients**

*Jacobo E. Mintzer, M. D., Linda Lewis, M. S. N., Leslye Pennypacker, M. D., Janet Pitner, Pharm. D., William Simpson, M. D., David Bachman, M. D., George M. Wohlreich, M. D., Alison Meeks, R. N., Steven Hunt, M. S. W., Kerri Hernon, B. S., Royce Sampson, M. S. N.*

### Special Article

- 377 Roderick Macdonald, M. D.: A Tribute**  
*Alexander G. Donald, M. D.*

### Editorials

- 383 Deserving Our Remembrance**  
*Charles S. Bryan, M. D.*

### Features

- 389 Alliance Page**  
**386 On the Cover**  
**353 President's Page**

### Association

- 387 Gray Matter**  
**369 SCMA Newsletter**

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## BICYCLE-RELATED HEAD INJURIES IN SOUTH CAROLINA CHILDREN: WHAT CAN WE DO TO PREVENT THEM?\*

NEAL J. THOMAS, M. D.\*\*

JANICE D. KEY, M. D.

WALTON L. ECTOR, M. D.

Injuries are a problem that physicians must deal with daily. While most doctors feel comfortable dealing with these injuries, prevention is something that is often overlooked. In most cases, prevention of the injury itself is much more beneficial than the treatment of the injury that results. Indeed, the old adage "an ounce of prevention is worth a pound of cure" is especially applicable to bicycle-related injuries. Cycle injuries, especially head trauma, are a common cause of emergency room visits, and it is well documented that wearing a protective helmet can significantly decrease morbidity and mortality from these accidents. Despite this, most children and adults still do not wear bicycle helmets.

This article will present the available national and South Carolina statistics and review the literature concerning the effectiveness and frequency of use of bicycle helmets. Also included

will be suggestions of ways in which physicians and other health care workers can promote helmet use among South Carolina children.

### STATISTICS

Bicycling is a very popular recreational activity in our society as evidenced by the approximately 96 million bicyclists in the United States today. However, it can also be a dangerous and even deadly one. According to Sacks, et al., there are 580,000 emergency room visits and 950 deaths annually in the United States as a result of bicycling injuries. Approximately one-third of all cycle related injuries involve head trauma. In fact, head trauma accounted for 62 percent of all deaths. The magnitude of these statistics is reflected in the fact that bicycling led to 905,752 head injuries and 2,985 head injury deaths between 1984 and 1988. While this is a problem with every age, children are at increased risk. Sack's study revealed children under 15 years of age accounted for 41 percent of head injury deaths and 76 percent of head injuries. Economically, this morbidity and mortality has an

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estimated annual cost of over \$1 billion, according to the Proceedings of the 29th annual meeting of the American Association for Automotive Medicine.

The prevalence of serious bicycle-related injuries is consistently documented in other studies. From December 1987 to February 1993, 61 trauma centers nationwide reported that 2,468 pediatric patients (aged 0-19) were admitted through trauma centers after bicycling injuries. This represented 8.6 percent of all trauma patients admitted to these tertiary centers. (Figure 1) This number is larger than gunshot wounds (5.3%), sports injuries (5.3%), beatings (2.8%), and motorcycle accidents (1.4%).<sup>3</sup> It must be emphasized that these numbers do not include children who did not require a trauma center or were pronounced dead at the scene of the accident.

In South Carolina, the problem is equally significant. In reviewing all E coded (coded by diagnosis) discharges (27.1% of all discharges) from 1987-1991, it was found that 128 head injuries or 7.8 percent of all children hospitalized for head injuries, were the result of bicycling accidents. The most common age groups were five to nine years old (43%), and 10-14 years old (38%). Males were three times more likely than females to have a bicycle-related head injury. Of these 128 patients, five patients expired and three were discharged to chronic care facilities.<sup>4</sup> In five years at the Medical University of South Car-

olina, South Carolina's only member of the Pediatric Trauma Registry, 57 children, or 9.6 percent of all pediatric trauma center patients, were evaluated for trauma that occurred from a bicycling injury.<sup>5</sup> Finally, according to South Carolina's Childhood Injury Reduction Project,<sup>6</sup> 48 children from South Carolina died from bicycling related injuries during the period from 1985 to 1989, with the majority being aged five to 14 years old.

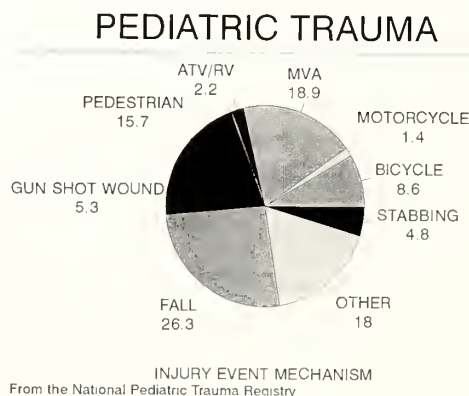
## EFFECTIVENESS

Many studies document the effectiveness of helmets. Mills et al<sup>7</sup> analyzed the mechanisms of energy absorption for frontal and side cranial impacts and found that a good helmet should protect the wearer for impacts up to 15 mph into a rigid flat surface. A landmark study by Thompson et al<sup>8</sup> concluded that riders with helmets had an 85 percent reduction in the risk of head injury and an 88 percent reduction in the risk of brain injury. Wasserman, et al, showed that helmet wearers experienced significantly fewer skull fractures (1% vs 11%) and facial soft tissue injuries (5%) vs 18%).<sup>9</sup> An additional study from Tucson, Arizona evaluated 298 patients over three years seen in a trauma center for injuries from bicycles involved in collisions with motor vehicles. The helmeted riders showed a significant decrease in the Injury Severity Score. The authors also pointed out that safer riding habits probably play a major role in injury severity.<sup>10</sup> This shows that a multifaceted approach is needed to combat these serious injuries.

The above statistics conclusively show that bicycling injuries are a major problem in South Carolina and nationwide. Since a majority of these injuries involved head trauma, increasing the number of riders who wear helmets properly will reduce the number of head injuries.

## HELMET USE

In 1986, the United States Cycling Federation rendered a decision that required all riders to wear helmets during officially sponsored races.



**Figure 1**

A survey showed that 80 percent of racers used helmets in training after this ruling. However, 64 percent of these racers had used helmets before the law was even in effect.<sup>11</sup> These experienced cyclists obviously feel that wearing a protective helmet is worthwhile, but the general population appears to feel differently.

Numerous reports have been published about the use of helmets in all bicyclists, and in children specifically. Prior to any interventions, the helmet usage ranges from two percent to 10 percent of all recreational riders, with most studies leaning toward the lower numbers. Weiss' 1985 survey showed 1.85 percent of elementary school children, zero percent of middle school children, 1.85 percent of high schoolers, and 10 percent of college students actually wore helmets.<sup>12</sup> Wasserman, et al., interviewed cyclists at the roadside and found that only 3.7 percent of children aged 11-19 were helmeted at that instance.<sup>13</sup> Otis, et al., estimated that the use of helmets in children is low, varying between two and four percent.<sup>14</sup> Of the 2,468 patients treated at trauma centers, the use of helmets was noted in only 31 (1.3%).<sup>3</sup>

In Charleston, South Carolina, in a survey taken by observation over a two-day period, 132 cyclists were observed, including 87 children. Although nine of 45 adults (20%) had helmets on, only two of the 87 children (2.3%) were wearing protective headgear. Although this is a very small sample population of cyclists, this 2.3 percent is not surprising and consistent with the results of most larger studies nationally. A review of the Medical University of South Carolina trauma registry statistics showed that of the 57 children seen for trauma, none had documented helmet use.<sup>5</sup>

Inadequate helmet usage in children may have several causes. DiGuiseppi, et al., looked at why parents hadn't purchased helmets for their children, and also why children didn't wear them. Fifty-one percent of parents stated that they "never thought about purchasing;" 29 percent thought the "helmet cost too much." Twenty percent thought that their

"child would not wear helmet;" and 12 percent stated that a "helmet is not needed." The latter group gave reasons including "child seldom rides, only rides around the neighborhood, and only rides where it is safe." The most common children's reason for not wearing helmets was "friends don't wear them" (28% of helmet owners and 25% of nonowners). In fact, they found that only 56 percent of all children who owned helmets actually wore them the last time they cycled.<sup>15</sup>

## WHAT WORKS TO IMPROVE HELMET USE?

The task to increase bicycle helmet use is a monumental one. Many strategies in many communities have been attempted with varied success.

The hallmark program was a community-wide campaign held in Seattle, launched in the summer of 1986. In this very successful campaign, various strategies were utilized to reach three goals: increase parental awareness, promote use by children, and reduce the financial costs of protective headgear. To increase parental awareness of the need for helmets, the program used multiple public service announcements, PTA presentations, posters, and bike tags. To promote use by children, a popular cycling group was featured to make it "cool" to wear a helmet. Multiple incentives, including sporting event tickets and french fry coupons, were given away at various times to bicyclists wearing helmets. And finally, to reduce the financial barriers to helmet purchase, multiple discount coupons for helmets to be sold at cost were distributed. In school-aged children, helmet use increased from 5.5 to 15.7 percent in 16 months after the campaign had started.<sup>16</sup> By three years, 33 percent of children in the Seattle area aged five to nine years old were wearing helmets.<sup>17</sup> Other avenues have been explored but have met with very limited success. These include school-based intervention<sup>18,19</sup> and emergency room counseling.<sup>20</sup> Therefore, most authors agree that physicians interested in increasing bicycle helmet use should direct their atten-



tion toward community based programs.

An excellent review article written by Dr. Jeffrey Brown, published in *Contemporary Pediatrics*, outlines the best steps to convince children and parents to wear bicycle helmets. His stepwise approach, includes forming a coalition, organizing support and funding, starting with smaller projects, and most importantly, branching out to involve the community. This article should be read by anyone interested in starting a helmet program in their community.<sup>21</sup> Table 1 is a list from this review which lists excellent places to get help in starting a campaign.

## LEGISLATION

Unfortunately, preventive health care on a mass scale has always required legislation in the past. Widespread acceptance of seat belts/restraints, infant car seats, and child-proof containers did not occur until mandated by law. Eventually, the use of bicycle helmets by children may also become mandatory nationwide. In fact, New Jersey passed a law that went into effect in July of 1992 that required bicycle riders and passengers under 14 years old to wear fastened and properly fitted helmets that met the standards of the American National Standards Institute or the Snell Memorial Foundation's 1984 Standard for Protective Headgear for Use in Bicycle Riding.<sup>22</sup> The state of Georgia recently passed a law that states "no person under the age of 16 years old shall operate or be a passenger on a bicycle...without wearing a bicycle helmet."<sup>23</sup> Howard County, Maryland, passed a similar law after two school aged boys died within a nine month period from injuries incurred during accidents on bicycles.<sup>24</sup> Dannebery and Vernick recently suggested a proposal for the mandatory inclusion of helmets in the purchase of all children's bicycles. However, until a similar legislative act is passed in South Carolina, an attempt to voluntarily increase helmet use is needed.

## SUMMARY

This review article demonstrates the clear

**TABLE 1**  
**WHERE TO GET HELP WITH BIKE HELMET CAMPAIGNS \***

Harborview Injury Prevention and Research Center  
325 Ninth Ave., ZX-10  
Seattle, WA 98104 1-206-223-3399

HEADstrong, c/o Ms. Sharon Thorson  
Injury Prevention Program, Colorado Dept. of Health  
4210 E. 11th Ave.  
Denver, CO 80220 1-303-331-8220

American Academy of Pediatrics  
Dept. of Publications  
141 Northwest Point Blvd.  
Elk Grove Village, IL 60009-0927 1-800-433-9016

Injury Prevention Resource Center  
Dartmouth-Hitchcock Medical Center  
Hanover, NH 03755 1-603-650-1780

TIPP information  
Sandoz Pharmaceuticals Corp., Bicycle Safety Dept.  
59 Rte. 10, Bldg. 701, 3rd Floor  
East Hanover, NJ 07936-1951 1-800-765-TIPP

Michigan Bicycle Helmet Advisory Committee  
Michigan Head Injury Alliance  
Michigan Dept. of Public Health  
3423 North Logan St.  
Lansing, MI 48906 1-800-537-5666

\*From Brown, J. *Contemporary Pediatrics*. 1992; 9(7): 56

need for helmet usage in children everywhere. The lives that could be changed and the money that could be saved from needless injury and death is substantial. As physicians and community leaders, we must all do our part to ensure the safety of children. This must be done by joining or initiating a local community campaign. Physicians need to become part of this preventive effort, both with their patients and by example. Hopefully, by following the suggestions of other programs already implemented throughout the United States and working together with the community, we can substantially decrease the number of needless bicycle injuries suffered by South Carolina's children. □

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# IN VITRO FERTILIZATION AND EMBRYO TRANSFER (IVF-ET) COMES TO THE UPSTATE\*

WILLIAM R. BOONE, PH.D.\*\*

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Assisted Reproductive Technology (ART), like many medical specialties, has changed dramatically in the last ten years. New techniques include in vitro fertilization and embryo transfer (IVF-ET), gamete intrafallopian transfer, zygote intrafallopian transfer and micromanipulation of gametes. The latest in ART is now available in the upstate of South Carolina through the Greenville Hospital System (GHS).

Although this successful program is new to the area, ART is not new. The first "test tube baby," Louise Brown, was born on July 25, 1978, in London, England.<sup>1</sup> The latest figures indicate that more than 4,000 babies were born in the United States and Canada following IVF-ET cycles initiated in 1991.<sup>2</sup>

The majority of our procedures to this date have been IVF-ET. In the following report we describe this IVF-ET procedure, associated laboratory techniques and the results obtained from our initial experiences with IVF-ET.

## MATERIALS AND METHODS

### *Patient Referral Base – Geographic Location*

Patients are referred from North and South

Carolina as well as other southeastern states. Of the 79 cases in this report, 68 originated from 10 different counties in South Carolina, eight came from North Carolina, and one case came from each of the following states: Georgia, Kentucky and Texas.

### *Patient Screening Procedure*

Couples are typically deemed eligible for IVF-ET when the following criteria apply: (1) absence or obstruction of both fallopian tubes that is deemed irreparable by current treatment methods, (2) extensive pelvic adhesive disease, (3) moderate to severe endometriosis, (4) compromised semen quality and/or (5) infertility of undetermined etiology where IVF-ET seems a reasonable means of attaining pregnancy.

### *Orientation for the Couple*

Couples electing IVF-ET attend an educational session. A nurse-coordinator presents information about the IVF-ET procedure, the medication protocol preliminary to the procedure and the effects (and potential complications) of the medications. The nurse demonstrates and supervises a practice session on mixing and administering the five hormone preparations and allows ample time for couples to have their questions answered. Each couple receives a workbook describing the procedure.

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TABLE I  
OVARIAN STIMULATION PROTOCOLS FOR IN VITRO FERTILIZATION PATIENTS

Protocol	Number of Patients	Mg. of Luprolide Acetate (LA)*	Amps of FSH†	Amps of hMG‡
With LA	67	15.0§	9.8¶	19.6**
Without LA	12	0	34.2††	9.5‡‡

\* Luprolide acetate is a gonadotropin releasing hormone agonist (Lupron)

† Ampules of follicle stimulating hormone (Metrodin)

‡ hMG is human menopausal gonadotropin (Peronal)

§ Range 5 to 28 mg

¶ Range 0 to 56 ampules

\*\* Range 0 to 38 ampules

†† Range 8 to 60 ampules

‡‡ Range 0 to 24 ampules

## TECHNIQUES

### *Controlled Ovarian Hyperstimulation*

The majority of IVF-ET patients are placed on a luprolide acetate (Lupron) regimen to down-regulate the pituitary gland prior to ovarian stimulation (Table 1). (The following is a general protocol and, thus, may vary among patients.)

Lupron, a gonadotropin releasing hormone (GnRH) agonist, is administered for approximately 10 days prior to the onset of menses and the start of the IVF-ET cycle. On Day 3 (Day 1 is the first day of menses) of the IVF-ET cycle, follicle stimulating hormone (FSH) and human menopausal gonadotropin (hMG) are given (Table 1). On Day 8, the FSH is discontinued. Serum estradiol ( $E_2$ ) concentrations and follicular sizes are carefully monitored, and human chorionic gonadotropin (hCG) is given when the serum  $E_2$  level and follicular sizes are appropriate. The hCG injection (10,000 units) is usually given when the  $E_2$  level falls within a range of 2500 to 3600 pg/ml and/or the largest follicle diameter averages 16 mm or greater on a two-dimensional picture as ascertained by vaginal probe ultrasound.

Patients who have responded poorly to past stimulation attempts receive a non-Lupron treatment which includes either hMG and FSH or FSH only (Table 1). In addition to the usual  $E_2$  and ultrasound determinations, daily luteinizing hormone and progesterone values are obtained to aid early prediction of impending

follicular rupture.

### *Oocyte Recovery*

For the majority of patients, intravenous (IV) analgesia is used to blunt the discomfort of the oocyte recovery procedure. The advantage of IV analgesia over general anesthesia, besides the post-operative advantages of being able to eat, drink and return home within hours after the procedure, is that patients are able to observe aspiration of follicles on the nearby ultrasound monitor.

Oocyte recovery is performed in a dedicated operating room (OR) (Figure 1) using transvaginal ultrasound guidance. An ultrasound probe, with a needle guide, is placed into the vagina. With the aid of the ultrasound machine, an image of the patient's ovaries is visualized on the monitor. Ovarian follicles in excess of 4 mm in size can usually be aspirated within 30 minutes using a 16-gauge double lumen needle (Cook OB/GYN, Catalog No. KOPSD-163301).

Follicular aspirates are passed to personnel in the ART laboratory (Figure 2) through a door connecting it to the OR. Oocytes recovered from the follicular fluid are placed into a 37°C incubator. The internal environment of the incubator is maintained at five percent  $CO_2$  in air with a relative humidity of between 95 and 100 percent.

### *Sperm Preparation*

Most semen samples are collected one to



Figure 1. Dedicated Operating Room (OR) for the Performance of Assisted Reproductive Technology (ART) Procedures.



Figure 2. State-of-the-Art Assisted Reproductive Technology (ART) Laboratory.

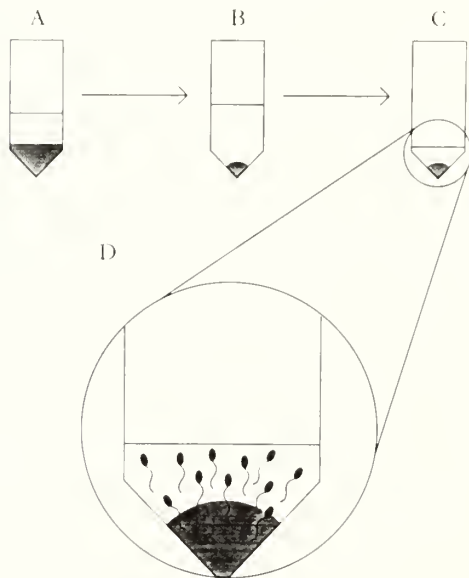


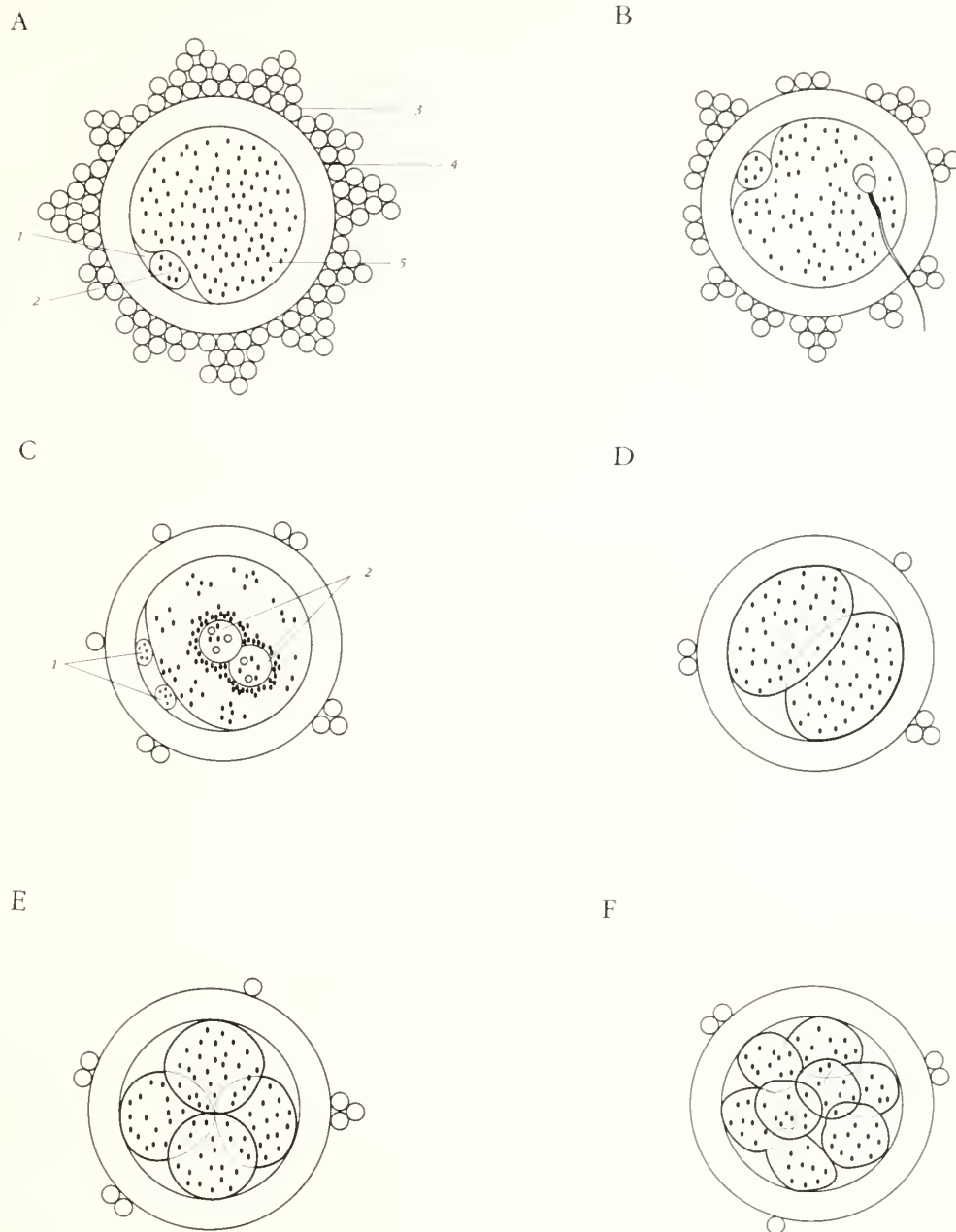
Figure 3. Sperm Washing and Swim-Up Procedure. (A) Culture medium is added to the semen. (B) Centrifugation forces the sperm into a pellet. (C) The sperm pellet is layered with a small volume of culture medium and incubated for approximately 60 minutes. (D) During incubation, the most highly motile sperm "swim-up" into the layer of culture medium which is then collected and used for insemination of oocytes.

seven hours after the oocyte recovery procedure has been completed. Samples are placed into test tubes and culture medium is added. The milieu is centrifuged ( $340 \times g$ ) to produce a sperm pellet at the bottom of the tube; the fluid portion is removed. The washing procedure is followed by a layering of the sperm pellet with a small volume of fresh culture medium. This layering procedure allows for the most vigorous sperm to "swim up" into the culture medium (Figure 3).

The culture medium-sperm mixture is incubated for approximately one hour prior to the removal of the supernatant. This incubation period allows sperm to capacitate, or alter their physiological state, a process that must occur before sperm can fertilize oocytes. A 10- $\mu$ l aliquant of the supernatant is diluted and analyzed for sperm concentration. After preliminary calculations, the appropriate volume of supernatant necessary to inseminate each oocyte with 100,000 or 500,000 motile spermatozoa is determined. The total number of sperm selected for insemination is dependent upon the qualities of the semen sample and the oocytes.

#### *Oocyte Fertilization and Embryo Development*

Oocytes are evaluated for maturity and exposed to sperm only after the appropriate stage of development is reached (Figure 4A, 4B). Once the sperm have been added to the oocytes, the gametes are left undisturbed for



**Figure 4. Anatomy of the Human Oocyte and its Developmental Stages After Fertilization with Human Sperm.** (A) Mature, unfertilized human oocyte: (1) Perivitelline space, (2) Polar body, (3) Corona radiata, (4) Zona pellucida, (5) Ooplasm. (B) Fusion of sperm with the oocyte. (C) Fertilized oocyte (zygote): (1) Polar bodies, (2) Pronuclei. (D) 2-cell embryo. (E) 4-cell embryo. (F) 8-cell embryo.

approximately 18 hours, after which time the cumulus cells surrounding the oocytes are mechanically removed and the gametes observed for evidence of fertilization. Fertilization is confirmed when two pronuclei are

observed within the cytoplasm. At this stage the cell is called a zygote (Figure 4C).

After confirmation of fertilization, the zygotes are placed in fresh culture medium and allowed to remain undisturbed in the



incubator until ready for embryo transfer.

### *Embryo Transfer*

Embryo transfer (ET) generally takes place at or near the 2-to 8-cell stage (Figure 4D, 4E, 4F) and requires no anesthesia. The patient lies supine, and a speculum is inserted into the vagina to visualize the cervix. A catheter containing one to four embryos (five to six embryos may be transferred to women 37 years of age or older) is passed through the cervix and into the uterine cavity. Once the catheter is in place, the embryos are released. The patient remains in the Trendelenburg position for an additional three hours before being discharged from the hospital.

### *Determining Clinical Pregnancy*

The Society for Assisted Reproductive Technology (SART) defines a clinical pregnancy as a rising beta-human chorionic gonadotropin ( $\beta$ -hCG) and the presence of a gestational sac(s) on ultrasound. Pregnancy rates are derived from the number of patients going to oocyte retrieval (whether or not any oocytes are obtained) divided into the number of patients that demonstrate a clinical pregnancy.

## RESULTS AND DISCUSSION

In May of 1993, SART published the pregnancy rate for the 21,083 oocyte retrievals initiated in the United States and Canada in 1991 (the most currently completed data).<sup>2</sup> The United States and Canadian pregnancy rate is 19.1 percent (4,017/21,083). The pregnancy rate for the 79 oocyte retrievals initiated by GHS in 1992 and 1993 is 27.8 percent (22/79).

The delivery rate for the United States and Canada is 15.2 percent (3,215/21,083). Considering the ongoing nature of many of our pregnancies, GHS expects a delivery rate of 24.1 percent (19/79).

At GHS, there were no meaningful differences between successful and unsuccessful couples with respect to patient demographics and prior reproductive history (Table 3). It is interesting to note that of the six women above 40 years of age, none became pregnant.

Couples achieving pregnancy had higher sperm counts and a greater proportion of motile sperm than nonpregnant couples (Table 4). Women who became pregnant had, on the average, higher estradiol levels on the last day of hormone therapy (2,460 versus 2,293 pg/ml), more follicles (13.6 versus 11.9), more oocytes retrieved (12.3 versus 10.2), and a slightly higher fertilization rate (81 percent versus 72 percent). Women who became pregnant also had a greater number of embryos available for transfer (5.7 versus 4.3) than nonpregnant women.

In those instances where embryos were returned to the patient, more were transferred in the pregnant group (4.1) than in the nonpregnant group (3.6). All couples in the pregnant group had at least two embryos transferred as compared to 10 patients in the nonpregnant group that had fewer than two embryos transferred (six patients – 0 embryos transferred; four patients – one embryo transferred). A higher proportion of embryos transferred to patients in the pregnant group were at a stage greater than four cells as compared to the nonpregnant group (63 percent versus 48 percent).

TABLE 2  
UNITED STATES/CANADA (US/C) AND GREENVILLE HOSPITAL SYSTEM (GHS)  
IN VITRO FERTILIZATION TREATMENT OUTCOMES

Group	No. of Retrievals	No. of Pregnancies	No. of Abortions	No. of Deliveries
US/C*	21,083	4,017 (19.1)†	802 (19.9)	3,215 (15.2)
GHS	79	22 (27.8)	3 (13.6)	19 (24.1)

\*Based on the most current data (1991) <sup>2</sup>  
†Numbers in parentheses are percentages

**TABLE 3.**  
**CHARACTERISTICS AND PRIOR HISTORIES OF PREGNANT VERSUS NONPREGNANT PATIENTS**

	<u>Pregnant</u>	<u>Nonpregnant</u>
Number of patients	22	57
Age 20-29 years (%)	8 (36)	12 (21)
30-39 years (%)	14 (64)	39 (68)
40 or older (%)	0 (---)	6 (11)
Mean height in cm (sd)*	164.8 (6.0)	165.9 (6.7)
Mean weight in kg (sd)	65.2 (13.3)	65.0 (14.0)
Percent ideal weight† (sd)	116.4 (24.7)	114.5 (25.8)
Previous pregnancy (%)	11 (50)	28 (49)
Previous ART cycle (%)	3 (14)	14 (25)

\*sd = standard deviation

†Actual body weight divided by the ideal body weight taken from a life insurance table.

**TABLE 4**  
**SELECTED VARIABLES FOR WHICH DIFFERENCES WERE PRESENT IN PREGNANT VERSUS NONPREGNANT GROUPS DURING THE INITIAL IN VITRO FERTILIZATION PROCEDURE**

	<u>Pregnant</u>	<u>Nonpregnant</u>
Number of patients	22	57
Sperm Preparation		
Prewash Sperm Concentration (million/ml)* (sd) †	89.2 (63.3)	81.2 (63.3)
Percent prewash TMS‡ (sd)	47.2 (49.2)	40.9 (42.5)
Postwash Conc (sd) (million/ml)	117.3 (84.7)	90.9 (80.8)
Percent postwash TMS (sd)	71.9 (69.4)	57.3 (51.0)
Estradiol Values		
Estradiol value (pg/ml) on day of hCG¶ (sd)	2459.8 (986.9)	2292.9 (1547.8)
Oocyte Fertilization and Embryo Development		
Number of follicles (sd)	13.6 (4.8)	11.9 (6.0)
Oocytes recovered (sd)	12.3 (4.7)	10.2 (7.5)
Percent fertilized (sd)	80.6 (17.6)	72.3 (30.0)
Number of embryos (sd)	5.7 (2.4)	4.3 (2.8)
Embryo Transfer		
Total number of embryos transferred (sd)	4.1 (0.7)	3.6 (1.2)

\*Data for nonpregnant group based on 49 patients; remaining 8 patients required donor sperm

† sd = standard deviation

‡ Total motile sperm

¶ hCG = human chorionic gonadotropin

Many couples with excess zygotes (fertilized oocytes) chose to preserve these cells for a future embryo transfer procedure using cryopreservation. This process involves exposing the zygotes to a cryoprotectant, placing the exposed cells into plastic straws and freezing them at a controlled rate to  $-180^{\circ}\text{C}$ . The straws and their contents are then submerged

in liquid nitrogen ( $\text{LN}_2$ ). Zygotes in this state of suspended animation may be stored for as long as three years. (These frozen cells were not incorporated into these data.)

## CONCLUSION

The Assisted Reproductive Technology (ART) program of the Greenville Hospital System

has now performed 79 IVF-ET procedures with a clinical pregnancy rate greater than the combined United States and Canadian average (27.8 percent versus 19.1 percent). Findings from GHS suggest that factors such as semen quality, estradiol concentration, number of oocytes recovered, percent fertilization and number of embryos transferred may influence the success of ART. □

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1. Steptoe PC, Edwards RG, Purdy JM: Clinical aspects of pregnancies established with cleaving embryos grown in vitro. *Br J Obstet Gynaecol* 87:757, 1980.
2. Society for Assisted Reproductive Technology: Assisted reproductive technology in the United States

and Canada: 1991 results from the Society for Assisted Reproductive Technology generated from The American Fertility Society Registry. *Fertil Steril* 59:956, 1993.

#### ADDENDUM

Since this manuscript was submitted, we have tabulated all data from 1993. During that year, 78 stimulated cycles were initiated with 70 (90 percent) resulting in a retrieval. A total of 64 of the 70 cycles (91 percent) underwent a transfer, and 25 (36 percent) women conceived a clinical pregnancy. A total of 22 of the 70 women (31 percent) have delivered or have an ongoing pregnancy.



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# SCMA NEWSLETTER

A PUBLICATION OF THE SOUTH CAROLINA MEDICAL ASSOCIATION

Joy Drennen, Editor

798-6207, in Columbia

Contributions welcomed

1-800-327-1021, outside Columbia

August 1994

## HIGHLIGHTS OF THE JULY 21 BOARD OF TRUSTEES MEETING

At the July 26 Board of Trustees meeting, SCMA President, O. Marion Burton, M. D., discussed his personal goal of promoting unity among the SCMA and specialty societies by establishing the Inter-Specialty Council to address legislative matters.

A statement of policy on ethical issues in managed care was approved by the board for publication in *The Journal*. ☐

## MEDICARE UPDATE

By now you should have received the August, 1994 *Medicare Advisory*. Included in this *Advisory* are complete directions on using the Automated Response Unit (ARU), Part B problem solving guide, who to contact with a problem, and other important information you should read carefully.

**Interest Rate Update:** The interest rate for overpayments is 13.0 percent effective July 1, 1994. This interest rate is applied to the amount due from a provider when a claim has been overpaid and is also applied to the amount underpaid by Medicare when additional benefits resulting from an appeal or hearing are not paid within 30 days of determination. There is a separate interest rate that applies to claims not paid timely. The new prompt payment interest rate is 7.0 percent effective for schedule payment dates of July 1 through December 31, 1994.

**ICD-9-CM Coding Update:** The ICD-9-CM coding system will issue an update for claims processed on and after October 1, 1994. Medicare will accept the new codes, as well as the current ICD-9-CM codes, from October 1 through December 31, 1994. You must use the new codes

for professional services billed on and after January 1, 1995 or your claim may be denied.

**Allergen Immunotherapy Billing:** CPT code 95165 has been removed from the list of procedures which must be billed hardcopy. You can now file claims for CPT code 95165 electronically!

**Non-Bilateral Services:** HCFA has recently clarified that certain non-bilateral procedures could be reported and paid on the same day. The codes which may be billed and paid for the same day service are: 11011, 11101, 17001, 17002, 17101, 17102, 17201, and 63048. HCFA has clarified that code 92543 is a single procedure which can only be billed once on any given date.

**Basic Billing Workshop:** Medicare has scheduled another Medicare Part B Basic Billing Workshop for October 12, 1994. Registration information is in the August 1994 *Advisory*.

(continued on page 2)



**Health Professional Shortage Areas Update (HPSAs):**

Effective for services rendered on or after August 1, 1994, the Lake City Service Area of Florence County has been classified as a HPSA. The Service Area includes the following Census Tracts:

- Census Tract 18: Lower half of Pamplico Division including the town of Pamplico
- Census Tract 20: Scranton & Northern Lake City
- Census Tract 22.01-22.02: Lake City West & East

Physicians must report modifier -QU in item 24d of the HCFA-1500 (12-90) claim form each line of service that was rendered in a HPSA located in Florence County.

HPSA incentive payments of 10 percent are calculated and issued once a quarter. *For additional information on HPSAs, please see your August and October 1993 Medicare Advisory.*

**Advisory Update:** Beginning in September, the medical directors office will publish all local draft medical policies in the *Medicare Advisory*. Physicians will have 45 days from the date published to respond to these proposed policies. Comments should be sent through the Carrier Advisory Committee representative for your specialty. ☐

## MEDICAID UPDATE

**Incentive Payment:** A one-time incentive payment will be issued to active primary care physicians who have served at least 100 Medicaid recipients during the Federal Fiscal Year 1993. The amount per client will vary (see outline below) depending upon the amount of recipients seen by each primary care physician. The report of primary care physicians who qualify for this one-time incentive payment was based on Early and Periodic Screening, Diagnosis and Treatment exams, office, prenatal and postpartum visits for claims from October 1, 1992 through September 30, 1993.

<u>Payment per Client</u>	<u>Number of Clients Served</u>
\$3.00	100 - 499
\$5.00	500 - 999
\$7.00	1,000 - 1,499
\$8.00	1,500 - or more

Office visit payments have not been increased due to budgetary constraints, so the purpose of this one-time payment is to offer some relief to primary care physicians for inflationary costs, and to represent the Finance Commission's monetary commitment to primary care physicians as we transition into a managed care system. All physicians who qualify for the incentive payment will be notified by letter of the payment amount and the number of clients served. A bulletin will be forthcoming with a detailed explanation.

**Revised Anesthesia Methodology:** Effective with dates of service on or after August 1, 1994, Medicaid will adopt Medicare's methodology for anesthesia team reimbursement. The unit rate for anesthesiologists and CRNAs, regardless of supervisory status, will be \$12.00. Any questions about the methodology change should be directed to the Department of Physician Services at (803) 253-6134. A *Medicaid Bulletin* is forthcoming with details. ☐

## DISEASE REPORTING

DHEC's ability to advise doctors of an increase in risk/incidence of disease occurrences in local areas is an important use of disease reporting data, which is directly applicable to a primary care physician.

South Carolina law requires medical care facilities or laboratories to report persons diagnosed with any disease DHEC identifies as a reportable condition. When reporting, consult the "South Carolina List of Reportable Conditions for 1994." To aid in reporting, DHEC plans to institute a 1-800 disease reporting line this summer that will operate 24 hours a day, 365 days a year. Until that time, reports can be made on the 1129 card by calling your local county health department, or (803) 737-4165 in Columbia. ☐



The SCMA Members' Insurance Trust, with 4,600 insureds, has contracted with Physicians Care Network effective August 15, 1994. Additional hospitals contracting with PCN include Colleton Regional Hospital in Walterboro and Providence Hospital in Columbia.

By now, all PCN participating providers should have received a letter requesting you to complete a business contact list. If you have any key contacts within the business industry, please complete the form and return it to PCN. This marketing strategy will greatly assist in recruiting businesses to contract with PCN.

*To request additional forms, please call Barbara Whitaker at (803) 798-6207, or statewide at 1-800-327-1021, ext. 226, or George O'Laughlin at ext. 251.* □

The Center to Prevent Hand Gun Violence and the American Academy of Pediatrics has unveiled a program to guide pediatricians, as well as other primary care physicians who treat families, in the task of approaching firearm injury.

STOP (Steps to Prevent Firearm Injury) is a free kit for physicians that contains a monograph, audio tape, patient pamphlets, office posters, and a bibliography. The kit advises physicians first to warn parents of the risks involved in gun ownership, and if they keep one at home, to empty it and lock it up.

*To order the STOP kit, write or call, Center to Prevent Handgun Violence, c/o D.M.A., P.O. Box 425, Bladensburg, MD. 20710-9974, (202) 289-7319.* □

### **SCIMER AWARDED GRANT TO PROVIDE CARE TO MEDICALLY UNDERSERVED CHILDREN**

The South Carolina Institute for Medical Education and Research (SCIMER) has been awarded a REACH OUT grant by The Robert Wood Johnson Foundation. The program - REACH OUT: Physicians' Initiative to Expand Care to Underserved Americans - is a national effort to mobilize physicians to enhance access to care for underserved Americans.

In alliance with the Department of Health and Environmental Control (DHEC) and other public and private agencies, the goal of SCIMER is to expand upon three existing public-private partnerships sponsored by DHEC entitled Child Health Initiatives by instituting partnerships between public health departments and private physicians in each of South Carolina's 13 health districts. These model initiatives provide care to children under the Medicaid program and vary in form to fit the needs of the local communities. SCIMER's program, PARTNERSHIPS FOR CHILDREN, will increase access to medical and preventive services while securing medical homes for Medicaid pediatric patients in an effort to provide continuity of quality medical care.

The REACH OUT grant consists of a one-year planning grant of approximately \$100,000. Based on progress achieved during the planning year, SCIMER will be eligible to receive a two-year implementation grant averaging \$200,000.

The Robert Wood Johnson Foundation also awarded Commu-I-Care (CIC) a \$100,000 grant to reduce administrative barriers for physicians and access barriers for patients. Specifically, the grant will be used to support new administrative functions of CIC. The grant will not replace current private or public funding for existing program activities. Instead, REACH OUT funds will be used primarily to hire new outreach staff to educate the CIC target population about the program and to work with local medical and DSS personnel, as well as to conduct enhanced media campaigns to increase public awareness about the program and to convene a Barriers Task Force of medical experts and community leaders to examine obstacles to access.

Twenty-two private physicians' groups in communities across the country were granted \$2.2 million to cultivate innovative methods of serving the medically underserved.



## SUPPLIES NEEDED IN GEORGIA FLOOD DISASTER AREA

The Medical Association of Georgia (MAG) is requesting donations of medical supplies to assist the Albany, Georgia area, which has been hard hit by recent flooding. Some supplies needed include:

- Ace bandages
- Antiseptic handi-wipes
- Aspirin
- Baby bottles
- Baby formulas
- Band-aids (all sizes)
- Bandages (all size)
- Bed pans
- Bug spray
- Chucks
- Depends (adults)
- Diapers (all sizes)
- DSGs
- Gauze pads (4x4)
- Glucometers
- Hospital beds (12)
- Hypo-allergenic tape
- Insulin syringes
- Latex gloves
- Oxygen tanks
- Sharp containers
- Stethoscopes (30)
- Thermometers
- Urinals

Materials may be sent to the MAG at 938 Peachtree Street, NE, Atlanta, GA 30309. For additional information, contact Priscilla Daves, (404) 876-7535.

## NINTH ANNUAL PRACTICE OPPORTUNITIES FAIR

To assist communities, hospitals and private practices in their efforts to recruit and retain physicians, the South Carolina Area Health Consortium (S. C. AHEC) will sponsor their 9th Annual Practice Opportunities Fair on August 19-20, 1994 at The Sheraton Hotel in Columbia, S. C. The fair is designed to help residents identify and evaluate practice opportunities throughout South Carolina. The first eight Fairs attracted over 885 residents and over 2,018 physician recruiters (hospital administrators, mayors, physicians, etc.).

Throughout the two-day event, residents from all of the state's teaching hospitals (and graduates of MUSC and USC in out-of-state residency programs) visit with representatives of various hospitals and communities to discuss locations, costs, assistance, and other variables associated with establishing a practice in South Carolina.

To register or obtain more information, please contact Mary Chesshire at (803) 792-9422 or Becky Seignious at (803) 792-4439.

## POSTER REQUIREMENTS

The following posters may be obtained by calling the appropriate agency, and where applicable, should be posted by all employers:

### S. C. Department of Labor, (803) 734-9600

- SCLD-1: SC Labor Law Abstract, Revised 6/90.
- SCLD-5SH-91: OSHA Poster covers the SC Occupational and Health Safety Act and supercedes all previous federal and state OSHA posters, including federal OSHA poster #2203. (Also available in Spanish).
- UCI-104: "Workers Pay No Part of the Cost for Job Insurance"
- UCI-105: "If You Become Unemployed"
- "Workers Compensation Works For You"

### U. S. Department of Labor, (803) 765-5981

- "Employee Polygraph Protection Act"
- "Federal Minimum Wage"
- "Notice to Employees Working in State and Local Governments"
- WH Pub. 1313: "Notice to Employees Working on Government Contracts"
- WH Pub. 1321: "Notice to Employees Working on Federally Financed Construction Projects"
- Migrant and Seasonal Employer Act

### Human Affairs Commission/EEOC, (803) 253-6336 or 1-800-521-0725

- "Equal Employment Opportunity is the Law"



# A NEW APPROACH FOR THE MANAGEMENT OF ACUTE PSYCHIATRIC DISORDERS IN ELDERLY DEMENTED PATIENTS\*

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Dementia is clinically described as a slowly progressive process of cognitive and functional decline.<sup>1</sup> In addition to their cognitive difficulties, demented patients will present with symptoms of paranoia, depression and severe agitated behaviors such as wandering, hitting, kicking, grabbing, screaming, intentional falling, performing repetitious mannerisms, or causing injury to self and others.<sup>4,6</sup>

These additional manifestations are particularly evident in the late stages of dementia, and the presence of psychiatric disorders, especially agitated behaviors, are major risk factors for nursing home placement.<sup>2</sup>

Currently, agitated behaviors associated with dementia are usually treated by the primary care physician or a non-specialized psychiatrist, and the treatment is predominantly pharmacologic with medications such as

haloperidol, benzodiazepines and carbamazepine.<sup>9,11</sup> This treatment approach, unfortunately, will prove beneficial for only a limited number of these patients with only approximately one third of patients with agitation responding to pharmacological treatment.<sup>11</sup>

## ALTERNATIVES TO CARE

In addition to a more traditional pharmacological approach, an alternative/complementary approach to the treatment of elderly demented patients with severe agitated behaviors is the use of behavioral/environmental techniques.

A useful concept in understanding this approach is "excess disability," which has been defined as "degree of functional impairment in excess of that expected due to the level of intellectual impairment caused by the dementing process."<sup>3</sup> Brody and collaborators demonstrated that when adequate attention is devoted to the patient's individual needs, clear potential for improvement is observed. As a clinical example, patients with severe language deficits will show their need to be toileted through increased agitation. The identification of this behavior as a request for toilet-

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\*From the Departments of Psychiatry and Behavioral Science, Medicine, Family Medicine and Neurology, Medical University of South Carolina, Charleston.

\*\*Requests for reprints should be sent to Dr. Mintzer at the Department of Psychiatry and Behavioral Sciences, 1st Floor Institute of Psychiatry, Medical University of South Carolina, 171 Ashley Ave., Charleston, SC 29425-0742.

ing (as opposed to random inappropriate violent behavior) through continuous observation of the patient's toileting schedules with appropriate staff response will ameliorate agitation and incontinence and improve the patient's functional abilities. Also, there is a diminished need for pharmacological interventions to control behavior.

Another important concept is Kurt Goldstein's "catastrophic reaction"<sup>8</sup> in which he suggests that the violent reactions observed in post-stroke patients are often related to environmental demands that exceed the patient's functional abilities (i.e., encouraging a conversation in a patient suffering from a language impairment). It is our clinical observation that the same principles apply to demented patients. In our experience, demented patients respond with agitated behaviors when stimulated or exposed to levels of demand which exceed their functional abilities. A clinical example will illustrate this point: a patient with a limited ability to focus attention who is placed for meals in a highly stimulating environment (family dining, loud conversation, television, noise) will develop violent behavior and will decrease food intake. When placed in a quiet environment the same patient will conceivably be able to focus attention on feeding. The patient is now able to eat, and the violent behavior vanishes. The management of the patient in an overstimulating or overdemanding environment (in relation to the patient's abilities) will trigger the onset of agitated symptoms. The continued presence of these elements in the patient's environment will sustain these behaviors and contribute to a sense of further deterioration, especially if the environmental triggers of these behaviors remain unidentified.

The authors consider agitated behavior to be a major cause of excess disability. This group proposes the reduction of excess disability, frequently manifested as agitated behavior, through a process that includes discovery of medical or psychiatric disorders. Appropriate interventions will reduce agitation and its negative consequences such as nursing home

placement. This approach is further enhanced by avoiding the onset of catastrophic reactions through the adaptation of the level of environmental demand commensurate to the level of the patient's cognitive and functional abilities.

The Behavioral Intensive Care Unit (BICU) at the Medical University of South Carolina has developed an environment where the level of care and observation will permit the etiological factors causing excess disability to be identified. The BICU treatment approach is based on the assumption that agitated behaviors are not random and are largely determined by undetected medical problems (such as asymptomatic urinary tract infections or pneumonia) or elements in the environment that trigger, sustain and extinguish the observed behavior.<sup>8</sup>

The elements triggering, sustaining or extinguishing agitated behaviors vary from patient to patient and from behavior to behavior. The BICU treatment strategy calls for a three week admission of uninterrupted, intensive observation and treatment. The treatment process consists of four steps: (1) identification of the agitated behaviors; (2) identification of patient, medical, environmental, and social factors responsible for the agitated behaviors; (3) design and implementation of both patient- and environment-directed intervention strategies; and (4) modification of the home environment and training for the patient's primary care giver to reduce the disparity between patient ability to adapt and the level of demand of the family and the home environment.

The intervention strategy is designed to modify the stimuli (environmental, medical, psychiatric) that cause agitated behavior while concurrently enhancing the patient's ability to adapt to provocative experiences. This may be achieved through environmental (physical, environment, family), medical (treatment of previously undetected disorders), psychopharmacologic, or psychotherapeutic interventions (behavioral).

After the intervention program has proven to be effective in the BICU, required modifications will be made to the home environ-

ment, and family members will be trained on environmental management techniques. Finally, the patient will be returned to the newly adapted home environment.

The ultimate outcome of this treatment strategy is the optimization of independent, non-institutional living for the elderly patient through the reduction of agitated behaviors. The BICU environment is used as a vehicle for transforming agitated behaviors into appropriate and even desirable behaviors.

## PRELIMINARY PROGRAM EVALUATION

The BICU opened in the Medical University of South Carolina's Institute of Psychiatry in February 1992. During the first nine months of operation, 55 patients were treated. The mean age was 79.13 with ranges between 57 and 91; 69.81 percent of patients admitted were female and 30.19 percent were male. The patient population was ethnically diverse with 77.78 percent of patients being White Non Hispanics, 20.37 percent African American, and 1.85 percent White Hispanics. An evaluation of marital status showed that 49.06 percent were married, 39.62 percent were widowed, 1.89 percent were single, and 9.43 percent were separated or divorced.

All patients admitted were diagnosed as suffering from dementia according to the DSM-III-R criteria (APA, 1987). The patients' level of cognitive deterioration was severe, evaluated using two different rating scales, the Mini-Mental Status Examination (MMSE)<sup>7</sup> and the Blessed Dementia Scale (BDS).<sup>1</sup> The average MMSE was 14.38 out of a possible maximum score of 30. Values below 17 on the MMSE demonstrated severe cognitive impairment. The BDS mean values were 7.4 with a maximum score of 17. Values over 4 on this measure demonstrated severe functional impairment. Approximately 40 percent of patients had an additional discharge diagnosis of major depression, and 60 percent had an additional diagnosis of organic delusional disorder according to DSM III-R criteria (APA, 1987). All patients were diagnosed as presenting with

severe agitation as defined by Cohen-Mansfield and Billig.<sup>4</sup> (In addition, all patients presented with at least three active medical problems.

The average length of hospitalization was 26.9 days and the mode was 21. An analysis of patient discharge dispositions shows that 75.93 percent of patients were discharged to their home, 7.41 percent were discharged to noninstitutional living environments (relatives or family members households), and 16.67 percent were discharged to nursing homes. A six-month follow-up telephone evaluation conducted by a psychiatric nurse showed that 92 percent of patients discharged home remained at their home six months after discharge. Eight percent of patients died during the six months following discharge. No patient was readmitted to the BICU or to any other psychiatric hospital during this period of time.

## CONCLUSION

Dementia is one of the most prevalent disorders observed in the elderly population. Psychiatric co-morbidity, especially agitation, affects a significant proportion of these patients, substantially increasing their risk for institutional placement. The BICU offers an innovative treatment alternative for the treatment of agitation and other psychiatric co-morbidity in demented patients and has demonstrated efficacy for prevention of institutionalization for these elderly individuals. □

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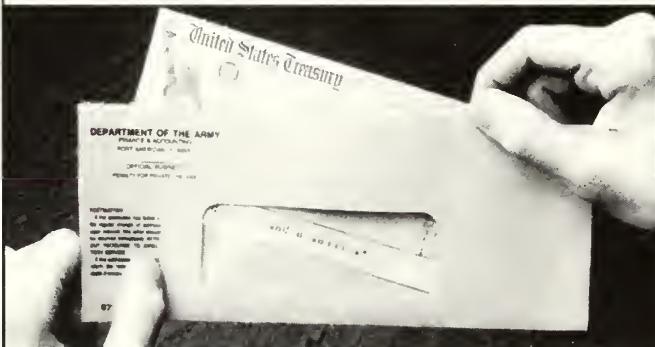
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# RODERICK MACDONALD, M. D.: A TRIBUTE

ALEXANDER G. DONALD, M. D.\*

Dr. William N. Adams-Smith was hired as Vice Provost for Health Affairs at the University of South Carolina, in July 1972. Public Law 92-541 authorizing the administrator of Veteran Affairs to provide financial assistance for the creation of eight new medical schools, which would be affiliated with Veterans Administration Hospitals (200 million dollars for the eight schools) was enacted in October 1972. Dr. Adams-Smith developed an application for the University of South Carolina. Richland Memorial Hospital, the William S. Hall Psychiatric Institute, the VA Hospital, and Moncrief Army Hospital signed formal affiliation agreements with the University so that the application could go forward. The South Carolina Legislature endorsed the establishment of the school with a commitment to provide the state's share of operating cost during the initial 10 years. The Veterans Administration awarded a 25 million dollar grant to the University for the development of a School of Medicine in June 1974.

A new VA Hospital had to be completed before renovation of the old VA Hospital could be accomplished for the permanent home of the medical school. Temporary offices, classrooms, and laboratories were found within the University. A "letter of reasonable assurance" was obtained from the Liaison Committee on Medical Education of the American Medical Association and the Association of American Medical Colleges in October 1974.

After an appropriate national search, a Dean was appointed. Within 92 days, he was abruptly relieved, and Dr. Adams-Smith became acting Dean. In December 1975, Dr.



Figure. Roderick Macdonald, M. D.

Adams-Smith, just as abruptly, resigned; and Dr. Francis Abel, chairman of the Department of Physiology and Pharmacology, was appointed Interim Dean of the new Medical School.

A Start Up Assistance Grant (\$420,000) was awarded by the National Institutes of Health. The Liaison Committee on Medical Education made a site visit in April 1976 and formally approved the admission of 24 students for the Fall of 1977. Dr. Roderick Macdonald was offered and accepted the position of Dean of the School of Medicine and Professor of Surgery (ophthalmology) in July, and arrived in Columbia to begin his duties in September 1976. As one of his conditions of acceptance, he was assured in writing that the Dean of the School of Medicine would report directly to the President of the University.

Why would Dr. Macdonald agree to a reduc-

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tion of approximately 50 percent in his salary in order to become Dean at a school in such turmoil and on the brink of collapse? There were at least three substantial reasons: (1) This would be one of the last new medical schools to be organized for many years; (2) The challenge of putting together a school along the lines of his personal convictions was appealing; (3) The combined opportunities of returning to his home state (some distant family connections had lived here since the late 1600s) and making a significant contribution to health care and the medical profession of the state were irresistible.

Dr. Macdonald's great grandfather, (also Roderick Macdonald) had been a physician in Scotland. His grandfather, Alexander Macdonald, came to the United States at 15 years of age, landing in Baltimore and from there coming to Blackstock, South Carolina (he had family in Fairfield County). He developed a thriving mercantile business, was postmaster and operated a dairy farm which had the first herd of pure bred Guernsey cattle in upper South Carolina. Dr. Macdonald's father was born in Blackstock. The eldest of three sons, he obtained his early education in a one room school. During high school, he taught several of the grammar school courses there. He attended the University of South Carolina from 1915 until his graduation in 1919 when he received the B. S. Degree.

After serving in the Army as a Second Lieutenant in the infantry, he entered the Medical College of South Carolina. Upon receipt of his M. D., he interned at the Roper Hospital. He practiced with Dr. Joshua E. Smith (an eye, ear, nose, and throat specialist) for about six months before going to Philadelphia for formal training in ophthalmology and otolaryngology. He returned to South Carolina and served as Health Officer in Fairfield county for several years before accepting a position as Chief of the Eye, Ear, Nose and Throat Service at the South Carolina State Hospital. Dr. Macdonald, Sr. was married in 1924. His wife was a native of Canada, and was a graduate of the nursing school at Roper Hospital. Dr. Rod-

erick Macdonald, Jr. was born in 1926.

During Dr. Macdonald's employment at the State Hospital, the family's living quarters were in the Babcock building. Dr. Macdonald, Jr. remembers the staff as an amicable group who worked cohesively in the comprehensive care of patients. In 1934, his father moved the family to Rock Hill where he joined the Fennell Infirmary as their specialist in eye, ear, nose and throat disorders. This infirmary was later purchased by the Catholic order of St. Philip. Dr. Macdonald then opened his own private office.

Dr. Macdonald, Sr. did graduate medical study in ophthalmology in London, Vienna, Glasgow, Edinburgh, Budapest, and Prague. He once told his son that added together he had the equivalent of approximately five years of additional training. In 1941, he spent two years at Yale University in their ophthalmology residency program. Dr. Macdonald, Jr. remembers the ambiance of Yale: its buildings, the people, and particularly Lord Halifax's 1941 graduation address. The senior Dr. Macdonald received his board certification in otolaryngology in 1939, and in ophthalmology in 1941. At the time, he was the only physician in the state who had both boards in these specialties. He served as president of the State Board of Medical Examiners, was President of the South Carolina Medical Association, was a member of the American College of Surgeons (Regent for the state), and worked closely with his old friend Dr. Kenneth Lynch to obtain a teaching hospital for the Medical College of the State of South Carolina. (He was always a devoted alumnus of that institution). He was also President, Secretary, Treasurer and Program Chairman of the state ophthalmological society, carrying the records of that organization in a shoe box until the membership voted to buy a briefcase for this purpose.

It was foreordained that young Roderick would be a physician like his father. He graduated from high school and attended Davidson College. Entering in the summer of 1943, he attended the regular academic school years and summer school before leaving to enter the



Medical College of South Carolina in 1945. He went back two summers in order to receive his Baccalaureate Degree in 1947. During his time at Davidson, he was judge of the court of control, a member of the Student Council, a member of Beta Theta Phi Social Fraternity, the Eumanean Literary Society, the staff of the school paper, and other campus groups.

In 1950, Dr. Macdonald, Jr. graduated from medical school. After a rotating internship at Baltimore City Hospital, 1950-1951, he did one year in a basic science course in ophthalmology and one year of ophthalmology residency (1951-53) at the Tulane post graduate School of Medicine and the New Orleans EENT Hospital. He was married in December 1951 to Helen R. Codington. They have five children. His wife's father was a surgeon and her two brothers are physicians. His wife has always supported his moves to various parts of the country as professionally indicated. The children always resisted, but liked it better once they were settled in the new location. Dr. Macdonald did a tour as a First Lieutenant in the Army Medical Corps 1953-55. During 1955-56, he served as Chief Resident in ophthalmology at Tulane and was asked by the Chairman (Dr. Allen) to be a Fellow for a year during which he would specialize in corneal transplant surgery and external diseases of the eye. He had always planned to go back to Rock Hill and practice with his father, but he enjoyed teaching so much that he decided to pursue a career in academic medicine.

Dr. Macdonald was offered an Assistant Professorship, and the position of Chief of the Ophthalmology Section at the Medical School at the University of Louisville, and served there from 1957-73. He became a Diplomate of the American Board of Ophthalmology in 1957 and served as an Associate Examiner for that board 1968-73. He was Associate Professor from 1961-65 and directed the sections conversion to an autonomous department in which he served as Professor of Ophthalmology and Chairman from 1965-73.

He also served as Associate Dean for Clinical Affairs from 1969-70, and Vice Dean from

1970-72. During these particular assignments, he concentrated on Medical administration. He served for four years on the National Institutes of Health Committee on Vision and Research Training, visiting many departments throughout the country to see what they were doing, assess their programs, and meet with their faculty. During his 16 years at Louisville, many students went into ophthalmology. Several graduates of that residency have become chairmen of other ophthalmology departments. He developed the eye bank and the Kentucky Lions' Eye Research Institute (a 2.5 million dollar eye research institute), but his greatest pleasure was in teaching residents and medical students.

In 1973, Dr. Macdonald was offered and accepted the challenge of moving to the Medical College of Virginia as Chairman of the Department of Ophthalmology. He quickly increased the revenue of this department, got the department out of debt, and became a director of the eye bank.

Upon coming to the University of South Carolina in 1976, Dr. Macdonald developed an effective organizational structure and recruited the essential faculty in the basic and clinical sciences. One of his first actions was the development of a mission and goals statement which was processed through the faculty of the School of Medicine, and the administration of the University; and was formally approved by the Board of Trustees of the University in June 1977. In essence it stated: "The School of Medicine of the University of South Carolina was established to improve the health of the people of the state of South Carolina through medical education, research, and the delivery of health care."

A major project was the development of an effective medical curriculum, which was under continuous study and revision. Temporary facilities had to be developed for use until the VA facilities could be renovated. This included not only classrooms and offices but also such things as laboratories, study areas, animal facilities and libraries, all of which are essential to medical education. There was

constant emphasis on public relations. A speakers' bureau and a newsletter which was mailed to every physician in the state were developed. An extensive continuing medical education program was developed.

The annual site visit of the Liaison Committee on Medical Education mandated a constant review of progress in the preparation of reports and answers for questions of the surveyors. Twenty-four students had been accepted with anticipation that approval might be obtained to start classes in the fall of 1976. This did not materialize. When the approval to start came in January 1977, there was pressure to begin a class immediately, but Dean Macdonald resisted and deferred to an August date. Twenty-four mature (many Veterans) students matriculated in August 1977, at an appropriate ceremony. This group was, and is, the Charter Class of the University of South Carolina School of Medicine. They had a particular feeling that this was *their* school. There was continuous input from them in the evaluation of the curriculum. Upon graduation, they gave the Dean a gold watch with the inscription "deepest regards, Charter Class 1981." It is understandably one of his most prized possessions. Thirty-six students were authorized to enter the school in 1978 and 48 students in 1980.

With the constant increase in the cost of medical education, about 70 percent of students require assistance. The bequest from the Corbit Estate (\$500,000) allowed for the development of low interest loans. The Columbia Medical Society and the South Carolina Medical Association developed scholarships. A Medical School Faculty Scholarship was developed. The establishment of the Todd Medical Endowment and the William Douglas Scholarships contributed significantly. Graduate incentive fellowships for minority students were developed by the state. There was a continuing search for funding to supplement state and federal appropriations. There was the establishment of a Chair in Hematology in memory of Frank L. Taylor, Sr. Since only five of eight potential schools developed, the

extra funds which would have gone to the other three schools were available for supplementary grants and an appropriate application was developed for a share of these funds. Funding for eight students to spend eight weeks in physicians' offices was obtained through the Duke Endowment. A Professional Practice Plan was developed and research grants, particularly in cardiovascular disease, cancer and diabetes were developed. (Each year there has been an annual increase in the number of research projects, the dollars in research grants, and the number of publications from the faculty).

A School of Medicine Honor Code and an advisement system were developed for students. Freshmen were assigned both faculty and sophomore advisors. Rising seniors were instructed concerning residency programs and how they might go about selecting the one that was most appropriate for them. Counseling on personal matters was available at the request of the students who knew confidentiality would be maintained. Liaison was developed with premedical advisors at all colleges within the state. Dean Macdonald's door was always open to students. He held regular luncheon meetings with the class presidents. There was a constant effort to recognize special accomplishments of faculty and students (teacher of the year, student banquet, etc). It was decided that the day before the University Commencement a ceremony honoring medical school graduates would be held on the historic USC Horseshoe (an ambiance conducive to the development of tradition). Faculty and student awards were presented. The oath of Hippocrates was administered by the Dean, and students received their academic hoods. The school applied for a chapter of Alpha Omega Alpha at Dr. Macdonald's behest, and approval for the establishment of such a chapter was given in December 1982. Dean Macdonald was selected to be the first faculty initiate in 1983.

Graduate programs were developed in anatomy, pharmacology, physiology, and microbiology. Departments of Radiology,

Ophthalmology, Preventive Medicine and Public Health were developed. Senior electives in clinical departments were developed in Florence, Anderson, and Greenville. After a special request from the University President to the Dean, the Department of Family Practice took over the operation of the Student Health Center of the University.

International activities included a visit in the fall of 1980 to the Peoples Republic of China (Shanxi School of Medicine) to form a continuing relationship and a visit to the Medical Research Institute of Alexandria in Egypt. (Their Dean was a visiting professor at USC in 1982.) Later Dean Macdonald arranged, through Baxter Laboratories, for the donation of hemodialysis equipment to Mrs. Anwar Sadat's Hospital in Cairo. An exchange of psychiatric residents between the Hall Institute and the University of Glasgow was developed.

The affiliation with the William S. Hall Psychiatric Institute and the William Jennings Bryan Dorn Veterans Hospital was most effective. Dr. William S. Hall and Mrs. Joan Kirschner were strong supporters of the school and constantly worked with the Dean for the betterment of both the medical school and their own hospitals. During the five-year period 1978-82, there was a 49 percent increase in applications for care at the Veteran's Hospital, with 96 percent acceptance, 9,400 inpatients, and 103,000 outpatients. Although the relationship with Richland Memorial Hospital was more problematic, the residency programs made major progress both quantitatively and qualitatively. The impact of the leadership of the academic faculty members of the school in the area was dramatic. A residency was developed in ophthalmology.

As one with strong convictions, inculcated from childhood, applies these convictions to the administration of an institution, the institution prospers and the individual bears the responsibility. Over time reactive feelings mount. The Commission on Higher Education felt that this school was established to produce family practitioners and could not appreciate

the need of all physicians to master a core of academic knowledge common to all specialties.

In 1982, the line of reporting from the Dean to the President was changed by the President so that the Dean reported to the Provost. This action was taken over the strenuous objection of Dean Macdonald as it represented a distinct change in his employment agreement.

All academic administrators (deans and department chairmen) serve at the pleasure of their superior, who may relieve them of that position at any time. This is in contrast to an academic position with tenure from which one can not be removed without major cause and for which there is always an appeal available.

Early in 1983, on a visit to the VA Hospital to survey the progress of renovations, Dr. Macdonald told his administrator that he did not believe that he would ever occupy the office that was designated for the Dean. In March 1983, shortly after he returned from Alexandria, Egypt with a signed affiliation agreement for faculty exchange and research, Dean Macdonald was called to the office of the Provost and without explanation, was ordered to "resign by 5:00 o'clock or you will be fired." Dr. Macdonald refused to resign and was relieved as Dean that evening without explanation. He retained his tenured professorship in ophthalmology and continued to be active in practice and in the education of residents and students (serving on the Admissions Committee of the School).

A committee of the Medical School faculty raised money and commissioned a portrait of Dean Macdonald by the eminent artist, Robert Bruce Williams, which was presented to the School of Medicine on April 27, 1984. It hangs in the Dean's Conference Room in the Administration Building of the School of Medicine.

In 1986, Dr. Dupont Guerry, III, a former Chairman of Ophthalmology at the Medical College of Virginia, invited Dr. Macdonald to join him in private practice in Richmond. Dr. Macdonald accepted and was engaged in active practice there until his retirement in 1992. At that time, he and his wife returned to



Columbia in order to be closer to their children and grandchildren.

Born with a strong medical heritage into a family with high moral and ethical principals, Dean Macdonald was taught early to be conscientious and to hold honored traditions in the highest regard. He made the most of learning opportunities, cultivating his abilities as a medical practitioner, educator, and administrator. Thus, he was uniquely qualified to be named Dean of the University of South Carolina School of Medicine. He developed order out of turmoil, he engendered an esprit de

corps among the faculty, staff and students, and he obtained full accreditation for this new medical school. Dr. Roderick Macdonald established a firm foundation upon which the school could grow-ultimately improving health care for all the citizens of South Carolina. □

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# Editorials

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## DESERVING OUR REMEMBRANCE

*As for man, his days are as grass;  
as a flower of the field, so he flourisheth;  
the wind passeth over it, and it is gone.*

Psalm 103: 15-16

*Life is short, the art is long...*

Hippocrates

The history of medicine holds a special place for those occasional physicians who died young but whose uncommon qualities and commitment exemplify our highest collective values. If we find in their deaths "that deep pathos in a life cut off in the promise of such rich fruit," as William Osler said of John Keats, we also find in their lives an affirmation of our own resolve to learn and practice medicine for the right reasons.

There was, for example, James Jackson, Jr., son of a famous Boston professor, who as a medical student in Paris meticulously recorded his cases of cholera and who rendered the first account of hereditary emphysema, only to die in 1834 of dysentery a few weeks after receiving his diploma. Jackson—the brilliant and dedicated student.

There was John Y. Bassett, rescued from anonymity by Osler in his essay, "An Alabama Student," who left his family and secure practice in Huntsville to seek out the best opinions of his day from the famous French professors, yet who died in 1851 of tuberculosis soon after returning to the United States. Bassett—the uncompromising practitioner.

There was Francis Weld Peabody, the young Harvard professor nearing the prime of life who, dying of cancer in 1927, left to a graduating class of medical students and to all posterity those stirring words, "The secret of the care of the patient is in caring for the patient." Peabody—the inspiring, idealistic teacher.

And there was Theodore Brevard Hayne, the enthusiastic young man from Congaree, South Carolina, whose interest in mosquitoes led him ultimately to take on yellow fever, who shortly after his marriage returned to Nigeria for unusually hazardous duty, and who in 1930 became the last martyred investigator of that disease. Hayne—the altruistic young physician-scientist.

The purpose of this editorial is to nominate two young physicians for this short but crucially important group: Susan P. Kuhlen and Oscar E. Glover, III.

Sue Kuhlen was caring personified. Before medical school, she was a nurse, and she seemed to make little or no distinction between the two roles. As a resident, she would do such things as take wheelchair-bound patients out of the hospital for a little fresh air beside the rose garden. As the minister put it at her funeral, she "was someone who knew how to love, and to be loved in return." Her ebullient personality and self-forgetfulness made her a favorite with everyone. But Sue was special not only for her compassion but also for her courage.

I remember being with Sue at her special appearance before the State Board of Medical Examiners, an appearance prompted by her particular disability. The executive director later commended her

for the candor and openness that you exhibited during your recent application for licensure. You have demonstrated a commitment



**Susan Piggott Kuhlen, M. D. (1956-1993)**

to the most cherished values of the profession.

For six months of residency training, she pressed on and cared mainly for others despite her relentless disease. Sue died as she had lived. Planning her own funeral, she told the minister: "Don't make it sad."

Oscar Glover was a role model in all respects. He was an example of what a young physician—or any physician—should be. Oscar loved to study medicine, but he loved life and people more. And he showed great promise. At South Carolina State, he was magna cum laude; at Meharry Medical College, he did so well that they wanted him back as a chief resident. He had that felicitous blend of knowledge from books, common sense, and human concern that make up the effective doctor.

Oscar's positive attitude and sense of humor made him a joy to be around. He made everyone's day just a bit brighter. He was never heard to complain even when circumstances were trying. I recall him expressing concern about taking a particular rotation that had been so notoriously difficult that other residents shied away from it. Having worked to improve it, I encouraged Oscar to try it which he did, to everyone's benefit. Oscar was someone to emulate.



**Oscar Ernest Glover, III, M. D. (1966-1994)**

Many persons called their deaths tragic. And certainly we grieve for their fine families, and for the people of Saluda and Orangeburg whom they intended to serve. But their deaths were far from tragic in the Greek sense—that is, tragedy understood as the result of a character flaw, a flaw evoking pity and fear. Neither had such a flaw—unless one considers too much caring or zest for life to be a flaw.

Sue Kuhlen had volunteered to draw blood from an HIV-positive hemodialysis patient in the emergency room. The patient's position in the room kept her from the needle dispenser, so she left the syringe on the stretcher momentarily while she disposed of the blood samples. When she returned to the room, a freak accident caused a small amount of blood to be injected into her abdominal wall, causing infection by the deadly retrovirus. Sue entered medical school that fall beneath a storm of controversy, but she quickly won the admiration of the faculty and fellow students. She completed medical school and six months of residency before the human immunodeficiency virus finally won. The minister concluded: "She taught us not only how to live, she showed us a way to die." Sue Kuhlen—a memorable lesson in caring and courage.



Oscar Glover, from the age of seven, was delighted by motorcycles. He was a careful cyclist who nearly always wore a helmet. But for some reason, he did not have it on that evening as he rode casually around Myrtle Beach. An unseen median in a darkened sidestreet resulted in his sudden death from closed head injury. Oscar was to have received his residency certificate the following week. Oscar Glover—a role model, a positive person who projected enthusiasm for medicine and all of life.

In his poem, “To an Athlete Dying Young,” A.E. Housman wrote:

Smart lad, to slip betimes away  
From fields where glory does not stay,  
And early though the laurel grows  
It withers quicker than the rose.

How different it is in medicine! A medical career resembles the rose, not the laurel; the preparation and nurturing are long and arduous, yet the bloom well worth the wait. How unfortunate, then, is the loss of two such bright young, energetic, committed, likeable, and promising young people. But they nevertheless contributed to the perpetuation of our collective ideals.

Jackson, Bassett, Peabody, Hayne, Kuhlen, Glover. In their lives are at least two lessons for us, the living. First, our younger contemporaries—like our older teachers and mentors—often have qualities that all of us would do well to imitate, to internalize. Second, such premature deaths remind us not only that life is indeed fragile and short, but also that it is our privilege and obligation to advance medicine in its best traditions.

Does reflection on their lives offer an even deeper meaning? Yes. If dying young cut off their opportunities to make sustained contributions to medicine, it also spared them the troubles and temptations that so often result from success. If we are honest with ourselves, we can see in their youthful idealism crystallized reflections of how we, too, once were. We can hear in the echoes of their memories those idealistic words that we, too, once told medical school admissions committees. Their memories should therefore prompt us to rededicate ourselves to lives of service. If we do this, they did not live and die in vain. Yes, they deserve our remembrance.

—CSB

# On the Cover:

## DANIEL LESESNE SMITH, M. D. 1877-1947 PRESIDENT, SCMA, 1928

D. Lesesne Smith was born in Berkeley County on July 28, 1877. He grew up in Mt. Pleasant and graduated from Porter Military Academy. He was in the first class to matriculate at Clemson, graduating in 1901. After earning his M. D. degree from the Medical College of the State of South Carolina in 1903, he practiced for several years in Newry and Great Falls, before moving to Spartanburg and limiting his practice to pediatrics.

His concern for children was great. He established the Spartanburg Baby Hospital, a charity institution as well as his private Infant's and Children's Sanatorium.

In a time of limited opportunity for continuing education for doctors, Dr. Smith founded in 1921 the Southern Pediatric Seminar in Saluda, North Carolina. This two-week postgraduate summer course was designed specifically for the general practitioner. Lectures

were given by some of the South's leading experts who paid their own expenses and donated their time to present the latest developments in pediatrics and obstetrics. The only expense to the student was a registration fee of \$25.

Lesesne Smith was also a loyal alumnus of the Medical College. He was instrumental in gathering funds to build the Alumni Memorial house, and in starting postgraduate seminars.

Dr. Smith was active in professional organizations both statewide and nationally. He was a devoted family man and was a "master of the art of living. He knew how to play and when to stop, how to make friends and how to keep them." He died of heart disease on July 7, 1947.

Betty Newsom

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# Alliance Page

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## THE 1994 AMA ALLIANCE ANNUAL SESSION DRAKE HOTEL, CHICAGO, JUNE 12-15, 1994

What a compelling, exciting, and rewarding experience for all of us! Attending were Donna Abercrombie, Kiki Sanford, Dee Jewell, Kakie Honig, Janelle Othersen, Gail Delaney, Hope Grayson, and Betty Hester. At the plenary session, "Focus: 94-95," the four major efforts of the AMAA for this year were characterized:

AMA - ERF - "One Voice for Medical Education." The medical student of today will be your physician tomorrow. Sixty million dollars has been raised for AMA ERF in the last 40 years, more than \$10 million in the last 10 years, and over \$2 million this year. South Carolina was recognized at this convention for increased AMA - ERF contributions.

Health Promotion - "One Voice for Good Health" - This project's focus will be on four public health issues: (1) One Voice to stop violence; (2) One Voice for healthy children; (3) One Voice for healthy teenagers; and (4) One Voice for effective parenting.

Legislative Affairs - "One Voice for Legislators" - as the AMA's partner in achieving effective health system reform, the medical alliance will urge all physician's spouses to remind state and federal legislators that no one is more qualified to speak to these issues than members of the medical community.

Membership - "One Choice - One Voice," Loud and Clear this Year. Membership is a team effort. "We're all in this together." We must have: (1) One Choice for impact on effective health reform; (2) One Choice for support of the medical family; and (3) One Choice for improving the health and well-being for the people in our communities.

At the opening session, the keynote speaker was Jeff Goldsmith, Ph. D., President of Health Futures, Inc. in Bannockburn, Illinois with the topic "Health Care Outlook." He said it is ultimately up to our spouses and to us how medicine will be practiced in our communities.

Maya Angelou - poet, educator, historian and actress - captivated convention delegates with her address: "Rainbows in the Clouds." Other speakers - Fred Barnes, Senior Editor of *The New Republic* in Washington D. C., gave his "View of Washington;" and Nancy Dickey, M. D., gave an AMA Board of Trustees update. Dr. Joseph T. Painter, M. D., President of the AMA, stressed that we must stay together, not divide ourselves. He highlighted the mini-internships program in Lexington County. Columbia Medical Society has also held mini-internship programs.

Betty Hester reported for the SCMA Alliance and we are very pleased that she will serve on the 1994-1995 National Promotions Committee. Hope Grayson will serve on the 1994-1995 AMAA AMA-ERF Committee. We were pleased to have Billie Brady, past president of the AMA Alliance, in our delegation. Barbara Tippo of Atlanta, Georgia was installed as the 1994-1995 AMA Alliance President. We enjoyed hearing her exciting message about adolescent health concerns.

Kiki Sanford (Mrs. H. Woodliff)  
President-Elect and Chairman of Delegates





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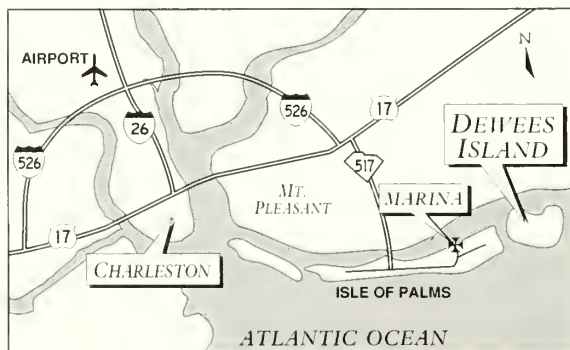
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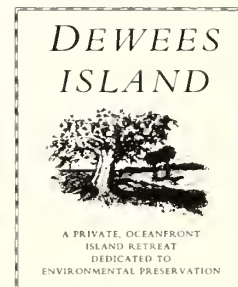
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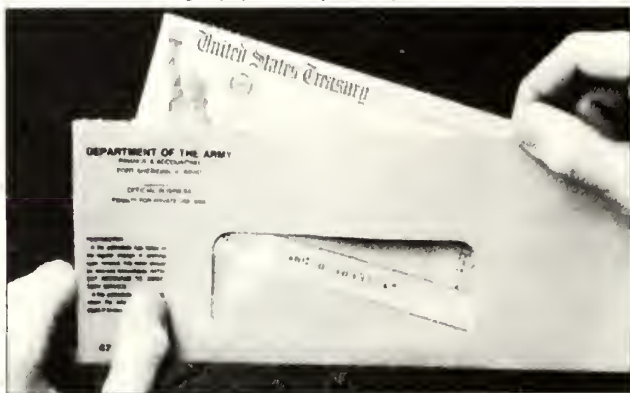
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## INDEX TO ADVERTISERS

B & B X-Ray .....	Cover 3
Anne M. Bowen, C. F. P. ....	Cover 4
The Cliffs at Glassy .....	352
Companion Technologies .....	351
Dewees Island.....	390
Doctors Hospital .....	391
Hancock Law Firm .....	Cover 4
Interim Health Care .....	Cover 2
Lexus of Charleston.....	368
Medical Protective Company .....	367
U. S. Air Force Active .....	382
U. S. Air Force Reserve.....	366
U. S. Army Active .....	376
U. S. Army Reserve .....	392
U. S. Navy .....	386
Winchester Surgical Supply Company .....	368





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# The Journal



OF THE SOUTH CAROLINA MEDICAL ASSOCIATION

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**SPECIAL ISSUE: ALZHEIMER'S DISEASE  
PART 1**

**GUEST EDITOR: CHARLES N. STILL, M. D.**

FRANCIS A. COUNTWAY LIB OF  
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# President's Page

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## A GOOD OFFENSE

Physicians in other states are experiencing a sudden and dramatic loss of patients, and it has nothing to do with their skills or care. These doctors are simply finding themselves closed out of health maintenance organizations, or having existing contracts terminated for no reason. The patients covered by these plans are "herded" to other providers who are often inconvenient and unfamiliar.

The name of the new game appears to be "market share and covered lives." An HMO with large numbers of enrollees is able to extract large discounts from some providers in exchange for volume. If the company is sold, each covered life in the plan is worth up to \$1,500. This sounds similar to the cellular phone industry mergers in which each potential customer is worth so many dollars. Free enterprise is to be coveted, but large for profit players cannot be allowed to have almost unlimited control.

South Carolina doctors and patients are just beginning to experience these calamitous currents that are devastating long term relationships in Texas, California, etc. Unless our team (organized medicine, hospitals and patients) wins, however, those of us in the Palmetto state will meet the same fate. Fortunately, our players are active. The American Medical Association (AMA) has gotten favorable response from Congress regarding key features of the Patient Protection Act which will assure patient and physician choices. The SCMA has been active in supporting state legislation that contains some aspects of any willing provider guarantees. The South Carolina Hospital Association (SCHA) is seeking antitrust relief to allow us to be more competitive in this new environment. Patients themselves are placing high priority on continuing to have the freedom to choose physicians and other providers. In summary, our *defensive* strategy is sound. It is often said, however, that the best defense is the *offense*. Having successful local managed care organizations in place when the entrepreneurs arrive will level the playing field. The SCHA is promoting the formation of community care networks which are locally governed by physicians, hospital executives and trustees. Your own *Physicians Care Network* has 2,200 physicians and 15 hospitals offering care to over 4,600 covered lives. We now need to build on this beginning and you must carry the ball.

First and foremost, if you are not a Physicians Care Network provider, *enroll today*. It costs nothing if you are an SCMA member. Second, if your hospital is not participating, insist that they join this partnership without delay. Third, identify local employers in your community who would benefit from this "middlemanless" network and contact Physicians Care Network (Barbara Whittaker or George O'Laughlin at 1-800-327-1021 or in Columbia 798-6207) and inform them of the potential employer client. The 4,600 covered lives could quickly become 100,000 or more, and your own managed care organization, Physicians Care Network, can be the best offensive weapon available for preserving high quality, cost-effective care in an environment that allows patient and provider choices.

O. Marion Burton MD

O. Marion Burton, M. D.  
*President*

## Contents

*Special Issue: Alzheimer's Disease: Part I*  
*Guest Editor: Charles N. Still, M. D.*

### 397 Introduction

*Charles N. Still, M. D.*

### 399 Alois Alzheimer (1864-1915) I. Contributions to Neurology and Psychiatry

*William L. Brannon, M. D.*

### 402 Alois Alzheimer (1864-1915) II. Dementia Before and After Alzheimer: Brief History

*William L. Brannon, M. D.*

### 404 Epidemiology of Alzheimer's Disease

*Caroline A. Macera, Ph. D., Youjie Huang, M. D., M. P. H.,  
G. Paul Eleazer, M. D., William K. Scott, M. S. P. H.,  
Carol B. Corrigan, R. N., P. A.*

### 407 Genetics of Alzheimer's Disease

*Ruth K. Abramson, Ph. D.*

### 417 Effect of Alzheimer's Disease on Communication Function

*Elaine M. Frank, Ph. D.*

### 424 Alzheimer's Disease and Caregiver Stress

*Lore K. Wright, Ph. D., R. N. C. S.*

## Features

### 436 Alliance Page

### 435 On the Cover

### 395 President's Page

## Association

### 429 CME Calendar

### 437 Gray Matter

### 413 SCMA Newsletter

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# The Journal



OF THE SOUTH CAROLINA MEDICAL ASSOCIATION

VOLUME 90

SEPTEMBER 1994

NUMBER 9

## SPECIAL ISSUE: ALZHEIMER'S DISEASE

GUEST EDITOR: CHARLES N. STILL, M. D.\*

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### PART 1: INTRODUCTION

Four score and seven years ago, Alois Alzheimer brought forth on the European continent the concept of a new disease process affecting the human cerebral cortex, dedicated to the proposition that though all men and all women would not be affected equally, all should be concerned. Alzheimer's address to the psychiatrists of Southwest Germany in the university city of Tübingen was all but forgotten for nearly 60 years. Then a remarkable discovery made independently by American and European researchers set off a renaissance of research on dementia, just 30 years ago. The pathology of Alzheimer's disease is the same, whether dementia begins at age 50 (presenile) or at age 80 (senile). To paraphrase the hyperbole of a contemporary commercial: This changed everything!

As the 'eighties began, large and small caregiver support groups across America united to form the national Alzheimer's Association, which seeks to encourage and to promote research into the cause and treatment of Alzheimer's disease. Next came the Decade of the Brain (1990-2000), signed into law by President George Bush on July 25, 1989. Among other goals, DOTB states that "the

causes of the dementias of childhood and adult life, and methods of early diagnosis and treatment, will be found in the Decade of the Brain." Having already reached the halfway point of the DOTB, we should not be discouraged if this lofty goal still remains beyond our reach in the year 2001.

In this issue of *The Journal*, Dr. William Brannon gives readers a concise biography of Alois Alzheimer and his impact on the neuropathological study of dementia at the beginning of the 20th century. Dr. Caroline Macera and her colleagues at the Statewide Alzheimer's Disease Registry offer a brief review of its epidemiology. Dr. Ruth Abramson details recent scientific advances in the genetics of Alzheimer's disease. Dr. Elaine Frank analyzes the effect of dementia on communication, memory and intellect during the progression of Alzheimer's disease. Dr. Lore Wright comprehensively reviews caregiver stress and strategies for coping with the "Thirty-Six Hour Day" experienced by those who care for persons with Alzheimer's disease.

Part 2 will emphasize newer approaches in the diagnosis and treatment of Alzheimer's disease and its most frequent clinical companion, multi-infarct dementia. We hope that readers of *The Journal* will find each of the

\*W. Jennings Bryan Dorn VA Hospital, Columbia, SC 29201.



papers in both parts useful and interesting.

As we welcome the new BMW assembly plant to South Carolina, let us not forget that Bavaria gave the world Alois Alzheimer 130 years ago, a generation before before the first automobiles appeared in Germany.

And Germany also gave us at least one-third of the European immigrants who were early settlers of South Carolina. Auf Wiedersehen! □

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## I. CONTRIBUTIONS TO NEUROLOGY AND PSYCHIATRY

WILLIAM L. BRANNON, M. D.\*

Alois Alzheimer was born on June 14, 1864 in Marktbreit, Germany, where his early education prepared him for university studies in Berlin and Tübingen. He was one of five sons born to a middle class Catholic family. His father, Eduard, was a public official. Little is written of his mother or what influences led him to study medicine; his brothers chose other professions. He earned his medical degree in 1887 in the University of Würzburg, and in the following year passed his state examinations with highest honors, earning the title of *Arztliche Approbation*.<sup>1,2</sup> He remained at the university for a brief period, working in von Kölliker's laboratory, before accepting a position in a public psychiatric hospital in Frankfurt. In the next 14 years under director Dr. Emil Sioli, he acquired the clinical skills for which he came to be so admired; his keen sense of inquisitiveness was honed and his appetite for clinico-pathological correlation generously fed. He was said to have had a natural scientist's attitude, to have loved nature, and to have possessed a talent for drawing anatomical figures. He did not care for the arts, nor did he care for politics. He loved teaching, once he began that career. His 20-seat laboratory classroom in Munich was always filled with students from many countries. To each he gave his careful personal attention during the laboratory sessions. Alzheimer was noted for his attention to teaching, for his ever present cigar and for the fact that he would frequently forget he had lighted one and light up another, so that the room would become filled with the aroma of

several burning cigars.<sup>3</sup>

In 1888, Wilhelm Erb (of Erb's Palsy fame), a friend of Alzheimer, called upon him to go to Algeria and escort a wealthy Kur who had developed tertiary lues. Alzheimer made the trip, and continued to treat the ill man until his death. In 1894, Alzheimer married the widow, Cecilia Geisenheimer.<sup>3</sup> Franz Nissl served as best man in the wedding. To that union three children were born; one subsequently married a psychiatrist who later associated professionally with Alzheimer. Cecilia died from an infection after seven years of marriage.<sup>1</sup>

The 19th century was a particularly prolific time for the clinical neurosciences.<sup>4</sup> In England, France, Germany and the United States, later to be recognized giants of discovery in brain diseases, were beginning their work. Figures such as David Ferrier, Hughlings Jackson and Charles Sherrington in England; Jean Charcot in France; Franz Nissl, Wilhelm Erb, and Emile Kraepelin in Germany; and William Hammond and S. Weir Mitchell in the United States were at work observing the natural history of neurological diseases, discussing the localization and physiology of the disease processes and defining the pathology. During the first half of the 19th century, there developed a difference of opinion between those who considered themselves localizationists and those who believed that it was not the particular localization of dysfunction that dictated the disease but rather what had gone amiss in cellular function was the prime issue in disease. Toward the middle of the century, concepts began to gel and turn in favor of the pathologists. In that setting Alois Alzheimer began his work.

As a student, Alzheimer came under the tutelage of R.A. von Kölliker, a histologist who was among the first to recognize that

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there are different kinds of cells in the brain, and that axons were derived from cells. His observations paved the way for Cájál to formulate the neuron doctrine for which he and Golgi were awarded the Nobel prize in 1906. Rudolf Virchow, Carl Weigert and Ludwig Edinger were also among Alzheimer's teachers in medical school, undoubtedly influencing his interest in pathology.<sup>5</sup> In addition to Sioli, Alzheimer was influenced in his formative years as a clinician by Franz Nissl, a clinical neuropathologist, with whom he retained a lifelong friendship and collaboration in research. Emile Kraepelin, the great psychiatric nosologist, provided additional motivation for Alzheimer's interest in relating diseases to their anatomic and histological pathology. After working in Frankfurt for 14 years, Alzheimer applied for a position as director of a state psychiatric hospital, and was rejected. Since becoming an administrator was Alzheimer's career plan, his rejection encouraged Alzheimer to seek an alternate career, and he became an academician under Kraepelin. Kraepelin had recruited Nissl to his institute in Heidelberg seven years earlier, in 1895. After Alzheimer's disappointment in 1902, he moved to Heidelberg; in 1903, he moved with Kraepelin to Munich, where he worked without pay since there was no formal position for him; he had received a modest family inheritance. Kraepelin nominated him for the title of *Privatdozent*. The move into teaching required Alzheimer to submit an original research work to establish his credentials (*venia legendi*) to become a junior faculty member, a process known as *Habilitation*, which he completed in 1904. After submitting his work, the *Habilitationsschrift*, Alzheimer was required to give a lecture, called the *Austrittsvorlesung*, to the faculty and students of the medical school. Following that, the lecturer was required to answer the questions of the faculty and students. If successful, as Alzheimer was, he was then allowed to give lectures to students and to charge tuition. The next level of academic accomplishment was the title of *Extraordinarius*, usually conferred after several years of

successful academic accomplishment. In some cases, a salary was offered along with the title. Alzheimer became *Extraordinarius* in 1908, but apparently did not receive a salary.

By the time Alzheimer became *Privatdozent* in Munich, he had published 20 papers in neuropathology. The work which earned his *venia legendi* involved a histological study of the differential diagnosis of progressive paralysis. That solidified Alzheimer's interest in cerebral cortical pathology which occupied most of his subsequent research efforts.<sup>1,2</sup>

It was Alzheimer's belief that psychiatric illnesses, including dementia and psychosis, could be best explained on the basis of neuropathology. Thus he devoted his major research and clinical interests to attempts to describe the neuropathology of each psychiatric illness. He set for himself a goal, never reached, of producing a book on the histopathology of psychosis. He prepared an early draft, but his untimely death halted the work. Throughout his career he retained an interest in clinical observation and treatment. He studied forensic psychiatry; he was particularly interested in juvenile delinquent behavior, which he thought might be hereditary, based on brain pathology.

At a meeting of South-West German Psychiatrists in Tübingen in November, 1906, Alzheimer presented a brief paper, *A Characteristic Disease of the Cerebral Cortex*.<sup>7</sup> In this paper he described a 51-year-old woman who began with a strong feeling of jealousy toward her husband; during the next four years she demonstrated progressive deterioration of memory and personality. She sometimes became delirious and at other times believed she was going to be raped by the doctors. She developed perceptual difficulties, and language abnormalities in writing and speaking, as well as in understanding. Motor skills were preserved for walking and for the use of her hands until near the end of her life, when she was completely apathetic, confined to bed in a fetal position. Alzheimer attended this woman when she arrived at the hospital in Frankfurt. Sioli provided the autopsy.



Alzheimer studied the brain and presented the findings: his description of neurofibrillary tangles is the hallmark of the presentation. Of the neurofibrils, he noted that they accumulated by forming dense bundles, and that they replaced the nucleus and cytoplasm of the cell. He also spoke of the deposition of a peculiar substance in the cerebral cortex (the Alzheimer plaque); that substance was later identified as degenerating neuritic processes and amyloid. What struck Alzheimer as so significant in that case is still unclear. In 1892 Arnold Pick had described a 72-year-old demented patient with focal abnormalities; in 1898 Redlich had described neuritic plaques.<sup>6</sup> Alzheimer believed he had described a new disease, or at least a new subset of dementia. He encouraged his colleagues to be attentive to subdivide well-known disease groups into smaller groups, each with its own clinical and anatomical characteristics.<sup>7</sup> It may be that Alzheimer's interest was whetted by the patient's age (51) at symptom onset. Agreeing that this patient indeed had a disease different from previously described cases of dementia, Kraepelin called the illness 'Morbus Alzheimer.' Kraepelin's influence prevailed, and the name stuck. The division between senile and presenile dementia remained for more than half a century, until modern neuropathologists clearly demonstrated that neurofibrillary tangles and senile plaques were the same whatever the age of the patient.

Alzheimer's subsequent work showed something of his diversity. He wrote about indications for abortion in mentally ill patients, and on epilepsy classification (1907). In 1910, he wrote of diagnostic difficulties in psychiatry; and later he wrote on the meaning of symptom complexes in psychiatry, and on the pathology of dementia praecox (1913). In addition, he published papers on Huntington's disease, juvenile degenerative diseases, tuberculous sclerosis, and amaurotic idiocy. Perhaps his *opus magnum* was a collection of papers on the pathological histology of mental disorders, edited in 1912.<sup>1</sup> In the clinic he was a master. Of the psychiatric case history he

wrote, "It seemed to me a list of judgments rather than symptoms; in many points, at the same time it is rather a reflection of the investigator's opinion and generally speaking, it is not a scientific document."<sup>1</sup>

In 1909 Alzheimer was honored with the appointment as editor of the prestigious *Journal of General Neurology and Psychiatry*. In 1912 he was given the Möbius Prize for excellence of research in the histology of psychosis; he was also he was appointed to the chair of psychiatry at the University of Breslau where he remained only three years. In 1912 he became ill with endocarditis, by report rheumatic or infectious. By 1915 renal failure developed and he died just before Christmas, at the age of 51 years. In addition to his description of Alzheimer's disease, he is also remembered for his contributions to the morphology of neurological disease; for his notion of the symbiosis between neurological and psychiatric illness; for his care of patients, for his meticulous work; and for his influence on the subsequent development of neuropathology in Germany and throughout the world. □

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# ALOIS ALZHEIMER (1864-1915)

## II. DEMENTIA BEFORE AND AFTER ALZHEIMER: A BRIEF HISTORY

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Dementia was not in Alzheimer's time a newly recognized disorder, though it was thought to be a process associated with aging. The ancient Greek judge Solon ( 500 BC) recognized the need to protect the mentally enfeebled by requiring that those persons making a will must not be influenced by "physical pain, violence, old age, or the persuasion of a woman."<sup>1</sup> The dementia of the aged was recognized by Plato; in *The Republic* Plato noted that the commission of certain crimes was excusable if committed in a state of madness, disease or under the influence of extreme old age.<sup>1</sup> In the first century BC, the poet Lucretius sang of dementia:

"...when the mighty force of years  
Their frame hath shaken, and their limbs  
collapse  
With blunted strength, the intellect grows  
dim.  
The tongue talks nonsense, and the mind  
gives way  
And all things fail, and all together go."<sup>2</sup>

Dementia thus was not so much considered a disease process as it was believed to be a part of the expected aging process. The Greek physician Galen, whose theses controlled medical thinking from the second century AD through the next millennium and a half, spoke of "morosis," meaning dementia. Galen defined "morosis" as "some in whom the knowledge of letters and other arts are totally obliterated: indeed, they can't remember their own names..."<sup>3</sup>

Shakespeare in his 1620 tragedy, *King Lear*;

has Lear recognize his progressing dementia thus:

"Does any here know me? This is not Lear.  
Does Lear walk thus, speak thus? Where are  
his eyes?  
Either his notion weakens, his discernings  
Are lethargied – ha, waking? 'Tis not so.  
Who is it that can tell me who I am?"<sup>3</sup>

Jonathan Swift described symptoms remarkably similar to Alzheimer's disease in *Gulliver's Travels* (part 3, Chapter 10). Of himself, eight years before his death in 1745 he wrote that he had entirely lost his memory; five years before his death he noted that he could not understand anything he wrote; and three years before his death, he was declared mentally incompetent and the management of his affairs given to caretakers.<sup>4</sup>

During the early 18th century, neuroscience began to emerge as the wisdom of Galen was questioned; for the first time in more than a thousand years autopsies became an acceptable practice. Concepts of dementia as a disease process did not change for the next hundred years. In the late 18th century, William Cullen offered a classification of nervous diseases in which senile dementia was recognized as a medical condition classified as *Vesaniae*, a group of diseases characterized as follows; "the judgement is impaired, without pyrexia or coma".<sup>5</sup> Benjamin Rush, Pinel and Esquirol each classified senile dementia as a disease. In the second quarter of the 19th century, Esquirol defined dementia clinically, "Senile dementia is established slowly. It commences with enfeeblement of memory, particularly the memory of recent impressions. The sensations are feeble; the attention, at first fatiguing, at length becomes impossible; the will is uncertain and without impul-

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sion; the movements slow and impracticable."<sup>1</sup>

In parallel with the clinical descriptions came neuropathological observation, and perhaps the beginning of the long-standing controversy in Psychiatry over organic versus non-organic explanations for psychiatric illnesses. Alois Alzheimer devoted much of his career to an attempt to define psychiatric illness in terms of its neuropathological substrate; and in that, he failed. He did, however, call attention to the fact that dementia was not the exclusive domain of the elderly; his influence led to the separation of pre-senile dementia, or Alzheimer's disease, from ordinary or senile dementia.

Current thinking about dementia has evolved into: (1) dementia as the primary disease process, i.e., Alzheimer's disease or (2) secondary dementia, due to cerebrovascular disease, e.g., multi-infarct dementia; or to certain infectious diseases, such as the acquired immunodeficiency syndrome; or to metabolic derangements such as thyroid dysfunction. Avenues for the study of the genetics of dementing illness are currently being opened; the ability to predict the development of dementia in individuals is at hand. With this come additional social and ethical issues, as well as medical issues.

The history of dementia continues: "Dementia is both a lonely personal experience and a shared worldwide crisis. We must work now by investing in basic research to find the roots of biologic solutions as well as by examining carefully what the goals of our care system should be. ...Addressing them will require not only intelligence but also integrity; answering them offers not only a better life for victims of dementia but also a more human life for all."<sup>6</sup> □

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# EPIDEMIOLOGY OF ALZHEIMER'S DISEASE\*

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## INTRODUCTION

Although recent estimates suggest that Alzheimer's disease currently affects three to four million persons nationally<sup>1</sup> and will affect close to 30,000 South Carolinians by the year 2000,<sup>2</sup> very little is known about the cause of this disease. Without a definitive diagnostic test, it is difficult to diagnose Alzheimer's disease, especially in the early stages. Most of the studies reported in the literature have used a case definition for Alzheimer's disease based on clinical symptoms, medical history, and exclusion of known causes of dementia. In spite of the diagnostic uncertainty, recent studies have provided insight into the disease process.

## PREVALENCE AND INCIDENCE

Two important indicators of disease burden and disease risk in the population are prevalence (number of existing cases) and incidence (number of new cases). Data from several studies generally agree that the prevalence of dementia increases exponentially with age. A general estimate of the population prevalence of Alzheimer's disease is about one percent among those aged 60-75 and about 10 percent among those aged 75 and older,<sup>3</sup> although some recent work suggests that the actual prevalence may be higher than these estimates.<sup>5</sup> As the population ages, the number of afflicted persons will increase sub-

stantially. The number of dementia cases in the United States is projected to increase by 34 percent between 1980 and 2000, although the population over age 60 will only increase by 19 percent.<sup>3</sup>

To study risk factors and trends, it is important to identify the incidence (number of new cases) of the disease. Because Alzheimer's disease is not easy to diagnose in its early stages, it is difficult to know exactly when the disease begins. Several studies that followed populations without evidence of cognitive impairment were, after several years, able to measure the incidence of cognitive impairment and then estimate the incidence of Alzheimer's disease. Based on these studies the incidence of Alzheimer's disease appears to increase exponentially among those between 60-90 years.

The duration of Alzheimer's disease (from onset of severe symptoms to death) can range from two years to over 20 years, with a median of about eight years.<sup>2</sup> Factors related to the duration of this illness include presence of other chronic conditions, access to medical care, and age at onset.

## RISK FACTORS

In the search for understanding and modifying the course of this disease, many potential risk factors have been studied. The most comprehensive analysis of potential risk factors was conducted by pooling 11 studies from six countries and re-analyzing the results.<sup>4</sup> Using this combined analysis and other relevant studies, the following sections briefly summarize the current scientific evidence on risk fac-

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tors for Alzheimer's disease.

*Family history:* Besides age, only family history of dementia in a first degree relative has been consistently associated with the development of Alzheimer's disease. There is some suggestion that a family history of Down's syndrome or Parkinson's disease is also associated with increased risk of Alzheimer's disease, but these findings are not consistent among the studies.

*Sex:* Prevalence studies have consistently reported a higher rate of Alzheimer's disease among women compared to men. Because of the longer life expectancy of women, this finding is not unexpected. The few incidence studies conducted have also found a higher rate of disease occurrence among women than men, but the difference is very small. No biological reason has been suggested as to why this disease would disproportionately affect women.

*Education:* While low educational attainment has been associated with the development of Alzheimer's disease, the nature of the relationship is not clear. It has been suggested that there may be a direct biological effect of education on the brain itself (increasing synaptic reserve), or there may be an indirect effect of education mediated through diet or other lifestyle exposures, or that this relationship may be spurious due to measurement inaccuracies.

*Ethnicity:* Between country comparisons may not be useful because many diagnostic tests are language-based and these measurement differences make the comparisons difficult. However, within the United States, no ethnic differences have been identified that could not be explained by educational attainment.

*Head injury:* In the pooling project, a strong association was found between head injury with loss of consciousness and the subsequent development of Alzheimer's disease. The mean time between the injury and diagnosis of

Alzheimer's disease was about 20-30 years, and this association was stronger in cases without a family history. Although not all studies have found an association between head trauma and Alzheimer's disease, this remains a promising area for continued investigation.

*Hormone replacement therapy:* While there is some evidence that cognitive function improves among postmenopausal women who are using hormone replacement therapy, several other studies have found no association. More work needs to be done in this area.

*Medical conditions:* Arthritis, headaches, and blood transfusions have been all weakly (and inversely) associated with the development of Alzheimer's disease, while thyroid disorders are positively associated with the development of Alzheimer's disease. Further study is needed to verify these relationships and to understand the mechanisms.

*Maternal age:* Risk associated with maternal age appears to follow a similar pattern to risk for Down's syndrome. Specifically, risk is highest among the youngest and oldest mothers (under 20 and over 40 years). While not all studies concur with these findings, this remains an active area for continued investigation.

*Psychiatric history:* While those with Alzheimer's disease or other dementia often have other psychiatric diagnoses, this may represent misdiagnosis of early symptoms of Alzheimer's disease. There is some evidence that treatment for depression may delay the onset of Alzheimer's disease, which could also mean that dementia was diagnosed later than it would have been had the person not been diagnosed with depression.

*Physical activity:* There has been some recent work suggesting that cognitive function improves with physical activity. How long this improvement persists or how this improvement would ultimately affect rates of Alzheimer's disease is not known, but physi-

cal activity remains an important area for future study.

*Aluminum and fluoride in drinking water, antiperspirant use:* Several mortality studies have suggested that exposure to aluminum in drinking water or through use of antiperspirants may be associated with dementia. Unfortunately, design considerations prevented these studies from answering this question effectively. Further work needs to be done in this area as well as in exploring the role of fluoridation on the absorption of aluminum.

*Unlikely risk factors:* Several studies have found no evidence to support the following as risk factors for Alzheimer's disease: birth order, animal exposures, stomach ulcers, antacid use, diabetes, allergies, tea and coffee consumption, and use of aluminum cooking utensils.

## SUMMARY

In summary, while only a few risk factors for Alzheimer's disease have been identified, several large collaborative studies of elderly populations are currently underway. As measurement techniques improve, the ability to diagnose this disease in its early stages will make the search for risk factors more feasible. □

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# GENETICS OF ALZHEIMER'S DISEASE\*

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Recently there has been a resurgence of interest in the inheritance of Alzheimer's Disease (AD) due primarily to application of molecular genetic techniques to the search for a gene or genes for Alzheimer's Disease. Many individuals are aware that Alzheimer's is present in more than one individual in more than one generation of their family. For some, the early onset AD among relatives promotes anxiety and raises the issue of what is their risk for developing the disease. For others, there may be only a sporadic case, i.e., one relative whom they can name, but the concern may be the same – is there a genetic predisposition to develop the disease, or is this a random environmentally induced event which will not recur? Is the genetic predisposition necessary, but not sufficient, for expression of AD? Did an at risk relative die prior to expression of the disease?

Heightened awareness of prevention raises the issue of the role of environment in ameliorating or preventing expression of AD. Recent research studies have provided clear evidence of the genetic diversity of AD, of the presence of genes which predispose to AD, and of the role of the environment, (both the external environment and the "genetic environment" defined by other genes). For clinicians, the main importance of these events is that identification of candidate genes provides insight into disease mechanism. For some genetic diseases, such as cystic fibrosis and severe combined immunodeficiency disease, there are now novel therapies.

## CLINICAL GENETIC STUDIES

Twin studies of AD provide unique informa-

tion. Monozygotic (MZ) twins are for the most part genetically identical, but the environment in which they are raised may differ. Dizygotic twins (DZ) are genetically and environmentally different. The study of identical twins gives information about genetic and environmental factors. A higher concordance rate (disease present in both twins) in MZ than DZ twins would be expected if AD were due primarily to genetic factors. The higher the discordance in MZ twins, the greater the role of environmental factors in expression of AD. Nee, et al.,<sup>1</sup> using the NINCDS-ADRDA Task Force using diagnostic criteria for AD, studied AD in twins ascertained over a period of two years through the ADRDA, Mothers of Twins Club, and physicians. Zygosity was determined by childhood photographs, family reports, red cell and HLA typing. Twenty-two twin pairs, in which one or both twins had AD, were diagnosed by NIH. AD, was confirmed by autopsy in four cases. The concordance rate for MZ twins in this study was 41 percent and for DZ twins, 40 percent. The data indicate that although genetic factors are present, environmental factors are important in the expression of AD. This study also showed that disease expression was often delayed in females.

Studies must take into account diversity of the age of expression of AD and whether the disease is familial or sporadic. Payami, et al.,<sup>2</sup> reported an association between the major histocompatibility locus (MHC) and AD. Patients (n = 54) and controls (n = 263) ascertained sequentially and rigorously diagnosed by the same team of physicians, were tissue typed for HLA A and B. An association between HLA A2 and AD was present in a subgroup of male patients, but not in male controls. There was no difference in female patients and controls. The frequency of HLA A2 was elevated in men with early onset

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dementia ( $\pm 60$  years). HLA A2 was present in 92 percent of men with early onset AD and only 44 percent of male controls. The association was primarily found in cases of sporadic AD (SAD) rather than familial AD (FAD). The importance of this finding may lie in the identification of a risk factor in the "genetic environment," i.e. an association with immune system regulation, that may enhance susceptibility to but not be sufficient for expression of sporadic early onset AD in men.

### MOLECULAR GENETIC STUDIES

In 1987, St. George Hyslop, et al.<sup>4</sup> reported genetic linkage in a few large families with early onset AD to chromosome 21q11.2 - 21q21, where the gene maps for a fragment of the amyloid precursor protein (APP). APP was found in brain amyloid plaques and vascular deposits associated with AD. In these families the gene is inherited as an autosomal dominant mutation, with a 50-50 risk of passing the gene to the next generation. This region of chromosome 21 is associated with Down Syndrome (DS), and the frequency of AD brain pathology is high in DS individuals who come to autopsy.

Schellenberg, et al.<sup>4</sup> and Tanzi, et al.<sup>5</sup> tested large numbers of AD families, sporadic AD cases, and controls to estimate the prevalence of mutation in the beta A4 domain of APP, including codon 717 mutation and two other mutations. Both studies found that these APP mutations are rare in both FAD and SAD, constituting three to four percent of all FAD, and about five to six percent of early onset FAD. Although a series of mutations in the APP gene identifies risk for early onset FAD, this rare mutation did not account for the majority of early onset cases or late onset FAD.

Schellenberg, et al.<sup>6</sup> studied a series of non-Volga German early onset kindreds in which AD was also inherited as an autosomal dominant gene. Linkage analysis gave highly significant lod scores for molecular markers on chromosome 14q24.3. This region of chromosome 14 contains candidate genes, including the c-FOS oncogene and HSPA2. The c-FOS

oncogene may be involved in transcriptional regulation of the APP gene. The product of the HSPA2 gene is a molecular chaperon which may be involved in protein assembly and degradation. Both of these genes play a role in injury response mechanisms in the central nervous system. Both can bind to a consensus sequence in the promoter region of the APP gene and could theoretically cause over-expression. The chromosome 14 markers account for approximately 70 percent of families with early onset autosomal dominant AD.

Currently identified genetic markers on chromosomes 14 and 21 account for 75 percent of AD in early onset families. These genes are sufficient for disease expression. This implies that there are different genes for the other 25 percent of early onset AD families.

Pericck-Vance, et al.<sup>7</sup> (1991) examined both early and late onset AD families with DNA markers on chromosomes 21 and 19. In late onset cases, there is difficulty in determining FAD because of the late onset, environmental influences, sporadic cases, and possible involvement of multiple genetic loci. An affected-pedigree member analysis was used to enhance this complex analysis. In late onset AD families, linkage was significant for chromosome 19q - 19q13.1.

Saunders, et al.,<sup>8</sup> based on the chromosome 19 findings, examined apolipoprotein E (APOE) in late onset FAD, SAD and controls. APOE, which is found on chromosome 19, binds amyloid beta peptide in cerebrospinal fluid with high avidity and specificity, and is found at autopsy in senile plaques; in neurofibrillary tangles; and in cerebral vessel amyloid deposits in individuals with FAD. They also found an association between the APOE E4 allele and late onset FAD and SAD. A large series of autopsy documented SAD patients also demonstrated a highly significant association with the APOE E4 allele. Saunders postulated that the APOE E4 alleles play a role in the metabolism of the amyloid beta peptide and may operate as a susceptibility gene for clinical expression of AD.

The APOE gene has a series of alleles,

including APOE E4. This means that individuals can have either one or two copies of APOE E4. Strittmatter, et al.<sup>9</sup> examined the DNA from 234 individuals from 42 families with FAD and additional samples from controls, describing a gene dosage effect. Almost all individuals who had two copies of APOE E4 developed AD. Their risk was eight times higher than the control population. Families with late-onset FAD have a single APOE E4 allele three times more frequently than controls.

Some cases of late onset FAD are linked to chromosome 19; in families with late onset FAD individuals who have two APOE E4 alleles are highly likely to develop AD. For late onset SAD cases, a single APOE E4 was present at a rate twice that in the control population. Thus, a single APOE E4 allele increases susceptibility to AD, but may not be sufficient to produce AD.

## CONCLUSIONS

The unfolding genetics of AD is a fascinating story which highlights the complexity of the interactions between genes necessary to produce illness, the environment, and other genes which may interact to increase disease susceptibility in the presence of a major gene. First, There is clear genetic diversity in familial AD. Genes on chromosomes 14 and 21 identify a majority of early onset familial AD. The Volga-German early onset AD families are not linked to chromosomes 14, 19, or 21; thus there is at least one other major gene which produces AD in autosomal dominant fashion. In some late onset FAD, there is linkage to chromosome 19, but further epidemiologic studies are needed to establish the prevalence of linkage to this site. Farrer, et al.<sup>10</sup> report that less than 50 percent of all AD is due to a major gene and that the major gene is not penetrant in all persons who survive to old age.

Second, at least two instances of "susceptibility genes," are known. There is an association between males with HLA A2 and SAD at early onset. Also, there is an association between the number of APOE E4 alleles in the individual and the likelihood of develop-

ing AD in late onset FAD and in late onset SAD. There may also be "susceptibility genes" which affect early onset FAD.

From twin studies it is clear that there are significant environmental factors which contribute to the development of AD. We do not know what these factors are. Does metal exposure, such as aluminum, in an individual with a genetic predisposition to AD constitute one such factor and head trauma another factor?

Genetic studies of AD are rapidly contributing to the understanding of the mechanism of the disease. However, great care must be taken when clinicians are asked by their patients to define an individual's risk for developing AD. At present, the APOE E4 gene should be viewed as a susceptibility gene, which may not be sufficient by itself for expression of disease. This highlights the need for definition of the role of environmental influences upon the expression of AD. The complexity of the pathogenetic mechanisms of AD rivals the complexity of the mechanisms of cholesterol metabolism and cardiovascular disease. Continuing genetic studies will contribute greatly to our understanding of mechanisms and eventual treatments of this devastating disease. □

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# SCMA NEWSLETTER

A PUBLICATION OF THE SOUTH CAROLINA MEDICAL ASSOCIATION

Joy Drennen, Editor

798-6207, in Columbia

Contributions welcomed

1-800-327-1021, outside Columbia

September 1994

## MEDICARE UPDATE

The September, 1994 *Medicare Advisory* has been issued. Please read this *Advisory* carefully as it includes fee schedule updates effective 10/1/94, guidelines for Cotigen implants and other important information.

**Professional Services Manual:** Medicare published the *Medicare Part B Professional Services Manual* last June. The manual is a comprehensive manual to assist SC Medicare providers in better understanding the Medicare Part B program. Look for updates to this manual in a special pull-out section in the November, 1994 *Advisory*. For the month of September only, Medicare is offering copies of the manual for only **\$20 per copy** (copies are usually \$40). To order, complete the order form on page 2 of the September *Medicare Advisory*. Orders must be postmarked by September 30, 1994 to receive this special price.

**Ophthalmoscopy (CPT codes 92225 and 92226):** Effective immediately for claims with services rendered on and after January 1, 1994, Medicare will reimburse CPT codes 92225 and 92226 separately when a general ophthalmologic examination (CPT codes 92002-92014) is performed at the same time. These codes represent unilateral procedures and can be filed for both sides when rendered on the same day. Medicare will adjust claims previously processed which are brought to their attention.

**Blood Count Procedures (CPT codes 85018-85027):** Some providers are incorrectly billing CPT code 85018 with codes 85022-85027. A CBC includes

hemoglobin so reimbursement for code 85018 is included in the reimbursement for code 85022-85027. You should **NOT** bill code 85018 with codes 85022-85027.

**Dialysis Claim Processing Update:** Medicare recently discovered that some ERSD claims were processed incorrectly. They are reviewing all dialysis claims filed for Hemodialysis (codes 90935 and 90937) and Peritoneal Dialysis (codes 90945 and 90947) which were incorrectly denied with action code "IJ." Medicare will adjust claims which were inappropriately denied. *You do not need to resubmit.* Medicare expects to have all claims corrected by 9/15/94. If your claims are not corrected by 9/15/94, please call the provider Service Center at: (803) 788-5568 (Participating Providers) or (803) 788-5569 (Non-Participating Providers).

**Provider Satisfaction Survey:** By now, you should have received a copy of the 1994 Medicare Part B Provider Satisfaction Survey. The purpose of the survey is to find out what **YOU** think of the job Medicare is doing. Your identity will remain anonymous. *Now's your chance to "say what you think."* Complete the questionnaire and return it in the postage paid envelope provided.

*If you need assistance with Medicare questions, please contact the provider Service Center at the above numbers, or call Cindy Osborn at the SCMA: (803) 798-6207, Ext. 238; or (800) 327-1021, Ext. 238.* ☐

**Provider Bulletins:** As of the beginning of this fiscal year (July 1, 1994), manual update bulletins will be numbered to assist providers with replacing pages in the proper sequence. For example, if you receive manual update #4 and have not received update #3, please contact your program manager at (803) 253-6134.

**Assistant C-Section:** A claims processing error has been detected for C-Section (59415) assistant surgeons. If your office has received any Error Corrections Forms (ECF) with error code 769 (assistant surgeon not allowed for this procedure) on

code 59415, please return them to your program manager to be recycled.

**New Provider Workshop:** This is a reminder that the Department of Physician Services offers workshops on a bi-monthly basis for newly enrolled providers or new provider staff. The next scheduled workshop is on October 5, 1994. The meetings are held at the Jefferson Square Office Building, 1801 Main Street, Columbia, SC, in the 2nd floor training room from 12:30 pm to 3:00 pm. Due to limited space, please notify our office at (803) 253-6134 in advance if you or your staff plan to attend or are interested in other available workshop dates. ☐

### **HCFA PILOT PROGRAM RE: CLIA**

The SCMA has been informed by HCFA that a pilot program will run in South Carolina and Utah during September concerning physician billing for lab services.

Nationally, there is significant discrepancy between the complexity of lab services billed for by physicians and the level of CLIA certification the physicians show on their CLIA number. The purpose of the program is to determine if HCFA and/or CLIA needs to do more education about their guidelines or change coding procedures.

There will be no punitive measures taken against physicians who receive letters from HCFA or the Medicare carrier under this pilot project.

*If you receive a questionnaire, please complete it as accurately and timely as possible.*

### **SEPTEMBER IS "WOMEN IN MEDICINE" MONTH**

AMA kicked off the fourth annual Women in Medicine Month in September to celebrate the achievements of women physicians and to promote increased participation and leadership opportunities for women in the profession and organized medicine. This year's theme is "Women Physicians on the Move."

Today, women comprise over 20 percent of all U. S. physicians and over 40 percent of medical students. The AMA projects that by the year 2010, women will make up 30 percent of the physician population. This growth is reflected in the increased participation and influence of women in organized medicine. The number of female AMA members increased by 24.4 percent over the past four years, making this the fastest growing membership segment. Equally important is the continuing growth of women physicians in leadership positions in the AMA and throughout the Federation. Women serve on the AMA Board of Trustees, as Presidents of their medical societies, and in the AMA House of Delegates.

*For more information on women in medicine, contact Phyllis Kopriva at (312) 464-4392.*

## PHYSICIANS CARE NETWORK UPDATE

Since the SCMA Members' Insurance Trust joined the Physicians Care Network (PCN) on August 15, approximately 200 physicians have enrolled in the network, bringing the total number of providers to 2,429.

At the present time, the following hospitals have contracted with PCN: Baker and Charleston Memorial Hospitals, Charter Hospital of Charleston, MUSC Medical Center, Roper Hospital & Health Services, and Trident Regional Health System (HCA Trident Regional Medical Center and Summerville Medical Center); Baptist Medical Center and Providence Hospital, both in Columbia; Carolinas Hospital System (Bruce Hospital, Florence General Hospital and The Women's Center) in Florence; Chester County Hospital; Colleton Regional Hospital in Walterboro; Edgefield County Hospital; Elliott White Springs Memorial Hospital in Lancaster; Lake City Community Hospital; Piedmont Medical Center in Rock Hill; and Newberry Hospital.

*For more information regarding PCN, please call Barbara Whittaker, in Columbia at 798-6207, ext. 226 or statewide at 1-800-327-1021, ext. 226.*

## POSTER REQUIREMENTS

Last month, the SCMA Newsletter featured a list of posters available from the SC Department of Labor, the US Department of Labor and the Human Affairs Commission/EEOC. Space did not permit publishing the following OSHA occupational safety and health recordkeeping materials:

**OSHA Form 200:** Log and Summary of Occupational Injuries and Illnesses (replaces OSHA forms 100 and 102). All employers with 11 or more employees, except those in certain exempt industries, must maintain and post this form annually.

**OSHA Form 101:** Supplementary Record of Occupational Injuries and Illnesses. This form, or a substitute containing the same information, must be completed for each case listed on OSHA 200.

*For further information or to receive copies of the above forms, contact the SC Department of Labor, Office of Public Information, PO Box 11329, Columbia, SC 29211-1329 or call (803) 734-9600.*

## VACCINE UPDATE

Vaccine Assurance For All Children (VAFAC), a new program administered by the State Department of Health and Environmental Control (DHEC), will begin October 1, 1994. Children eligible to receive vaccines under VAFAC include:

1. Children enrolled in Medicaid
2. Children who do not have health insurance
3. Children who are American Indian or Alaskan Native.

Children who have health insurance which does not cover vaccine may receive VAFAC-provided vaccines at federally qualified health centers (community/migrant health centers) and rural health clinics.

The following vaccines will be available:

DTP, DT, Td, DTaP, DTP/Hib, Hib, OPV, IPV, Hep B (for children born in or after 1992, and for chil-

dren through age 5 years in the household of a Hep B carrier), and

MMR (for age 12-15 months, preschool, and college freshmen).

DHEC will ship all vaccines directly to your office.

An allowable administrative fee for the vaccines will be \$10.00 per visit if associated with another billable visit. For a shot only visit or in association with a non-billable visit, the allowable administration fee will be \$20.00 per visit. Medicaid will publish a special bulletin concerning billing procedures for the administration fee.

*In order to participate, you must enroll with DHEC as a VAFAC provider. For more information, please call 1-800-27-SHOTS.* □



## PUBLICATIONS/VIDEOTAPES AVAILABLE

The AMA has published *Culturally Competent Health Care for Adolescents: A Guide for Primary Care Providers*. This book describes a framework for understanding and developing cultural competence, which is the sensitivity, cultural knowledge, skills and actions that enable physicians to work effectively with patients who are from cultures different from their own.

To order copies, call the AMA Order Department toll free at 1-800-621-8335 with Order #OP017894. The price for AMA members is \$7.50 per copy, and \$11.50 for non-members.

The SCMA Risk Management Program offers a new videotape on loan entitled "Communication: The Caring Touch." Produced by the Pennsylvania Chapter, American College of Emergency Physicians, this 10-minute videotape features situations applicable to all specialties.

To view the videotape on loan, call SCMA Headquarters and ask for Pam Taylor, Ext. 229.

## AMA PURCHASE LINK

AMA Resources, Inc., launched its AMA Purchase Link program nationally September 1. This national launch follows a four-state regional rollout of the program, which provides AMA member physicians with a direct access to Henry Schein, Inc. (HSI), the largest nationwide telesales distributor of medical, surgical and pharmaceutical products to physicians' offices.

The arrangement will provide AMA members with guaranteed low pricing on every item; no fees, purchase commitments, or minimum orders; free shipping for AMA members; a broad selection of more than 18,000 items in stock, including medical, surgical pharmaceutical, laboratory, and office

supplies; quality services, including responsive sales representatives and same-day shipping on most orders; and a reduction in time required to manage clinical supplies.

The national launch of the program follows a successful regional rollout in Iowa, Indiana, Ohio and Wisconsin during the past seven months. Beginning September 15, South Carolina office-based physicians who are AMA members will be contacted and referred to a toll-free hotline (1-800-772-4346) through which they can contact HSI directly. Watch for a mail-out from AMA Resources, Inc. later this month. □

## CAPSULES

Four distinguished South Carolinians were honored last month by the SC Chapter of the American Academy of Pediatrics during its Annual Scientific Meeting at the Hilton Resort, Hilton Head Island.

*Ms. Linda D. Price*, Division Director of Children's Rehabilitative Services, was named Child Advocate of the Year for her contributions to the health and well-being of SC's children and for her achievements on behalf of these children.

*Owen Ravenel, MD, and Bachman Smith, MD*, both from Charleston, received the 1994 Career Achievement Award. Their careers paralleled one another; both were revered by their patients for their many years of faithful service.

The prestigious President's Award was presented to *C. Morrison Farish, MD*, for his outstanding service to the chapter, its activities and the children of the state.

# EFFECT OF ALZHEIMER'S DISEASE ON COMMUNICATION FUNCTION\*

ELAINE M. FRANK, PH. D.\*\*

"Well, this the place, this here, that is the man going. She is getting stuff. Don't you see? And the two boys, momma both the same and here. The man is here, boys can see but hiding, um, um, and this is the man – yes."

This example of the speech and language of a patient with dementia due to Alzheimer's Disease (AD) is fluently formed but yet not communicative. What is the source of the problem, and how do the communicative attempts of patients with AD provide insight into progression of dementia?

Within the last decade, the disproportionate growth of the elderly segment of the population has increased the research focus on the aging patient. Studies have examined overall differences in speech and language function across the life span. New insights have emerged concerning the communication changes associated with normal aging and with dementia. This understanding of communication function is critical for accurate assessment of the geriatric patient. A well designed evaluation can determine if specific areas of loss are present, provide a profile for differential diagnosis between dementia and other potential causes of cognitive and communicative losses, establish a baseline of functioning, and obtain information that is helpful in counseling patients and caregivers .

## NORMAL DEVELOPMENTAL SPEECH-LANGUAGE CHANGES

\*From the Department of Speech-Language Pathology and Audiology, the University of South Carolina, and W. J. Bryan Dorn VA Hospital, Columbia, SC.

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Normal aging effects may be subtle and gradual in onset. Effective communicative functioning is dependent on abilities to use language, memory and intellect.<sup>1</sup> Certain of these critical processes may diminish across decades but significant losses are uncommon except in the very old or secondary to the presence of disease.

*Language Changes:* Language has three basic components: form, content and use.

Form is the word production and syntactic linking of words according to linguistic rules. Aging has very little effect on this syntactic structuring of language. Obler<sup>2</sup> reported that subjects from 60-80 had no loss of syntactic knowledge and produced sentences with elaborate grammatical style similar to subjects in their 30s. Results of investigations of age differences in complex receptive tasks varies ranging from a mild linear decline across decades<sup>3</sup> to no age-related differences.<sup>4</sup>

The content component of language includes the topic involved in an utterance and the vocabulary to express or interpret meaning. Elders may have some reduction in speech encoding and mild inferential processing losses, reducing receptive information transmission. Any sensory deficits such as hearing or visual loss have an added negative impact on receptive processing. In contrast, elders continue to increase content potential with a gradually expanding semantic concept and vocabulary lexicon store.<sup>5</sup>

Use is the pragmatic implementation language including the ability to recognize the rights, obligations, and expectations underlying maintenance of conversational discourse in varying settings.<sup>6</sup> Elders may be slower to process and produce language, resulting in less efficient discourse comprehension, con-



frontation naming, reading or writing.<sup>1</sup> However, the elderly retain pragmatic abilities, including the skills of effective exchange of information through conversation.

*Memory Changes:* Memory models have established a paradigm to include working, recent, and long-term memory. Working memory contains information which is in immediate use by the individual. Recent memory contains data related to events within the recent past; some data which will then be processed for long-term storage. Within long-term memory, several types of information are stored in either procedural or declarative knowledge. Procedural knowledge stores the rules or procedures for activities. It is the "knowing how to" information. Declarative knowledge stores concepts, names of objects (lexicon) and episodic (event) memories.<sup>7</sup> Procedural memory is well maintained in elders as is most general declarative information. Within declarative knowledge, studies have found mild deficits in quick access to the lexicon and in recent memory for events or episodic knowledge.<sup>1</sup>

*Changes in Intellect:* General knowledge or crystallized intelligence is retained. Elders may have difficulty in timed tasks resulting in a mild loss of information processing and timed problem-solving ability.<sup>8</sup>

## COMMUNICATION PATTERNS IN AD

Dementia has an insidious and progressive effect on communication function. Table 1 summarizes the effects of dementia on language, memory and intellect during the early, middle and late stages of AD.

The semantic loss of word finding ability is well established in the literature of early dementia.<sup>5</sup> Initially, the loss of naming may be noticeable only for infrequently used words or words with multiple or subtle meanings. Fluency naming tasks are particularly difficult for patients with AD. Fluency naming includes two tasks: (1) confrontation naming: requesting a patient to name an object presented and

(2) generative naming: naming items in a category or beginning with a specific letter. Patients with AD have increased response times and reduction in the number of total responses on all types of naming tasks.

Critical semantic attributes for distinguishing related objects may be unavailable to individuals with AD. A horse may be identified as a dog or a table as a chair. These examples demonstrate a loss of the critical features defining these related objects.<sup>9</sup> Syntactic and grammatical performance remains relatively intact until the very late stages of dementia. A reliance on this syntactic ability has been found in mild and moderate dementia subjects. When requested to freely name any item related to a stimulus, normal elders produce a paradigmatic response or a word within the same grammatical category (e.g. boys yield a response of girls). In subjects with mild and moderate AD, significantly more syntagmatically related responses (e.g. boys yield a response of play) are produced. In the middle stage, the semantic word-finding difficulty increases to include confusion of more common object names and, in the late stage, jargon productions.<sup>10</sup>

Reduction in naming ability and lexical access impacts on picture description and story telling. In the early stage, AD patients are able to relate picture information but require more time to communicate ideas. Productions reflect a loss of detail and are more concise than productions of normal elders. During the later stages, productions are impoverished or often not available to the dementia patient.

Pragmatic skills are resilient in the early stage of dementia; however, patients have difficulty in maintaining conversational responsibilities. Remaining on a single topic too long, repetition of ideas or slowness to initiate responses may make the conversation partner uncomfortable but communication is achieved. More abstract forms of conversation including humor, sarcasm, indirect statements and idiomatic nonliteral frameworks may be difficult. Pragmatic skills continue to deterio-



**TABLE 1**  
**EFFECTS OF DEMENTIA ON COMMUNICATION, MEMORY AND INTELLECT**

EARLY STAGES		
Language		
Form	Sounds: Grammar:	Used Correctly. Generally Correct.
Content	Expressive	May omit meaningful noun in a sentence, "tip of the tongue" behavior and reduced naming ability. Reducing vocabulary. Divergence from topic. Reduced ability to produce a series of meaningful sentences. Retention of social phrases. Comprehension of new information difficult.
	Receptive	
Use	Pragmatics	May talk too long on a topic, repeats ideas, may be unable to initiate conversation at appropriate time, able to answer most questions. Difficulty understanding humor, sarcasm, indirect and nonliteral statements.
Memory	Working/Active Declarative Long-Term	Shortened span. Less efficient retrieval and encoding of new information. Poor episodic memory and degraded conceptual memory.
Intellect		Reduced problem solving ability.
MIDDLE STAGES		
Language		
Form	Sounds Grammar	Used Correctly. Complex sentences difficult to understand. Sentence fragments common.
Content	Expressive	Difficulty thinking of words in a category and anomia in conversation. Vocabulary reducing and difficulty naming objects. Frequently repeats ideas or forgets topic. Reliance on topics from past. Empty language. Retention of social phrases.
	Receptive	Increased difficulty comprehending new information or complex topics. Diminished comprehension of written and spoken language.
Use	Pragmatics	Poor sensitivity to context and communication partners. Concrete. Misses humor and nonliteral language meanings. Knows when to talk and recognizes questions. May fail to greet. Usually does not self correct.
Memory	Working/Active Declarative Long-Term	Prominently Impaired. Deterioration of conceptual memory and lexical access.
Intellect		Significant and obvious deficits.
LATE STAGES		
Language		
Form	Sounds Grammar	May be used correctly but error not uncommon, some patients mute. Lack of comprehension of complex forms. Frequent sentence fragments.
Content	Expressive	Poor vocabulary and marked anomia. May produce jargon or bizarre meaningless sentences. Unable to produce sequence of related concepts. Repetition of words and phrases.
	Receptive	Lack of comprehension of new information. Most meaningful are past events.
Use		Unaware of surroundings, context, rules of conversation or communication partners. Little meaningful use of language.
Memory	Working/Active Declarative Long-Term	Severe deficits. Severe deficits.
Intellect		Severe deficits.

Adapted from Bayles, K.A. in Chapey, R. *Language Intervention in Adult Aphasia*, 1994. Williams and Wilkins.

rate in the middle stage. Poor sensitivity to context and communication partners is common. Patients may fail social greeting and may not be able to correct errors made in conversation. In the late stage of dementia, pragmatic skills are severely affected and little meaningful use of language is observed.<sup>11</sup>

### DIFFERENTIAL DIAGNOSIS

Differentiating developmental aging changes from dementia requires assessment of memory and semantic functions. Although episodic memory may be less efficient in normal elders and in individuals with dementia, the degree of loss is more severe in AD. Working memory and semantic functions are significantly more impaired in patients with dementia. Even in the early stage of dementia, retention of new information, social aspects of topic conversation and anomia are characteristic.

A state of severe stress in depression can result in pseudo-dementia. Ruling out depression as well as other reversible physiologic conditions is essential. Unlike individuals with dementia, during the communication assessment depressed patients may exhibit self-deprecatory comments, statements about feeling sad or helpless, flat affect, failure to try, and inconsistency in performance.

Dementia can be differentiated from other diseases specifically affecting language, including aphasia, primary progressive aphasia, vascular dementia and right hemisphere disorders. The onset of aphasia immediately after a cerebrovascular accident (CVA) contrasts sharply with the gradual onset of dementia. Aphasia results in a more focal loss of language with a preservation of primary memory and intellect, while dementia has a more diffuse etiology affecting cognitive function.

Recently a progressive form of aphasia has been described. This syndrome, primary progressive aphasia, has an insidious onset and is defined as a specific language loss with preservation of cognitive intellect and, therefore, no dementia. Patients with progressive aphasia can perform nonverbal cognitive

skills within established normative levels.<sup>13</sup>

The vascular dementia secondary to multi-infarcts presents with a step-wise progression secondary to vascular changes but may otherwise be quite similar to AD dementia. The progression through the language characteristic of the three stages of dementia may correlate with vascular changes.

A nondominant hemisphere CVA, usually in the right hemisphere, has characteristics of language loss often attributed to dementia. Patients with a right CVA may have reasoning difficulties, orientation problems, attention deficits, visuoperceptual problems and pragmatic communication problems. These patients have a focal history of quick onset and do not decline in function over time. The right hemisphere group also does not have the severe linguistic losses profiled in dementia.<sup>14</sup>

### COMMUNICATION ASSESSMENT

The communication assessment provides a baseline of function. In progressive disorders like dementia, baseline data can be compared to periodic testing scores, to determine changes in function. The evaluation procedure must include a thorough case history from the patient and the caregivers. The clinical evaluation of the patient's ability to provide case history information offers an opportunity to assess the integrity of the intellectual, memory and communication systems. Caregivers may provide information from environmental situations relevant to diagnostic decisions. After determining the premorbid literacy level of the patient, a standardized dementia rating scale is administered. Several scales are listed in Table 2. The scales contain a number of verbal items and screen the performance level of patients in verbal fluency, orientation and naming.

Traditional assessment tools for the evaluation of adult language have included tests such as the *Boston Diagnostic Aphasia Examination*<sup>14</sup> and the *Western Aphasia Battery*.<sup>15</sup> These assessment batteries were designed to sample behaviors differentiating patients with a focal aphasic disorder from individuals with

TABLE 2  
DEMENTIA RATING SCALES AND AUTHORS

Dementia Rating Scale	Author
Blessed Dementia Rating Scale	Blessed, Tomlinson & Roth, 1968
Clinical Dementia Rating (CDR)	Hughers, Berg, Danziger, Coben & Martin, 1982
Dementia Rating Scale (DRS)	Mattis, 1976
Functional Assessment Stages (FAST)	Reisberg, Ferris, deLeon & Crook, 1984
Global Deterioration Scale (GDS)	Reisberg et al., 1982
Mini-Mental State Examination (MMSE)	Folstein, Folstein & McHugh, 1975

normal elderly language function. These tools, although helpful in the assessment of moderate and severe dementia cases, are not designed to assess the subtle changes presented by the mild AD patient.

In 1993, Bayles and Tomocda published a speech language assessment battery specifically designed to sample the speech and language of individuals with dementia, the *Arizona Battery for Communication Disorders of Dementia* (ABCD).<sup>16</sup> This battery contains 14 subtests that evaluate five areas, linguistic comprehension, linguistic expression, verbal memory, visuospatial skills and mental status. Two subtests, delayed story retelling and word learning, have been found to distinguish performance of individuals at risk for dementing diseases and normal elders. Mild AD patients achieve significantly poorer scores than normal controls and moderate dementia subjects are poorer than mild dementia subjects.<sup>1</sup> The delayed story retelling and word learning subtests are particularly susceptible in patients with dementia, as the tests rely heavily on memory and new learning. A clinical assessment of the communication and problem solving strategies that patients utilize during the completion of the test is clinically revealing. Patients may exhibit lack of organizational ability, verbal access or frustration in memory tasks.

A second test, the *Functional Linguistic Communication Inventory* (FLCI) provides an assessment of the quality of functional communication.<sup>17</sup> Abilities tested include naming, greeting, answering questions, writing, sign and picture comprehension, word reading and comprehension, following commands, and the

ability to reminisce and to participate in a conversation. This inventory is particularly helpful in developing Functional Maintenance Programs (FMPs) for patients and families. FMPs provide the family with realistic goals and intervention strategies to maximize communication, maintaining meaningful interactions between patients and caregivers.

### THERAPEUTIC INTERVENTION

Therapeutic intervention seeks to maintain the patient's communication level and to reduce stress and communication frustration for caregivers interacting with the patient. Several techniques are available to encourage continued exchange of information with the patient with AD. Patients and care givers are encouraged to interact in functional activities requiring communication exchanges. Table 3 lists a number of techniques to increase the comprehension of the AD patient during communication. Primarily suggestions focus on simplification of communication utterances, reduction of reliance on memory and maintenance of patient dignity during communication interactions.

### SUMMARY

Dementia has a significant impact on communication abilities. The profile of communication skills in dementia differ from the profiles of developmental aging and focal organic disorders. Normal elders may have some episodic memory losses and slower reaction times as aging progresses. Focal language disorders (aphasia and right hemisphere disorders) will affect specific language functions without affecting intellect and memory. Communica-



**Table 3.**  
**CHANGES TO IMPROVE COMMUNICATION**

<b>Form</b>	
Simplify syntax	<p>Subject-verb-object sentences are least demanding on memory.  Example: <i>John hit the ball.</i>  <u>not</u> <i>The ball was hit by John.</i></p> <p>If a dependent clause is used, place it after the main verb.  Example: I saw you when I went to the game  <u>not</u> <i>When I went to the game, I saw you.</i></p> <p>Reduce length of sentence.  Example: I boughtt bread. I mailed the letter.  <u>not</u> <i>I bought bread and mailed the letter.</i></p>
Caregiver uses slower than normal rate of speech	Normal speakers use 160-200 words per minute. Slowing speech to below 120 words per minute reduces the number of concepts to be processed and the memory load for the patient.
Use several modalities to present information to patient.	Seeing hearing and tactile modalities provides more information than any single modality alone.
Limit number communication partners at one time.	Patients do best one on one rather than in groups.
Use pleasant accepting vocal tone	Patients with dementia can still process emotional characteristics of speech. Demeaning tone or baby talk rhythm is not appropriate.
<b>Content</b>	
Talk about here and now.	Eliminate dependence on episodic memory by conversing about something patient can see or hold.
Simplify vocabulary	Use more common words.
Reduce use of pronouns	Pronouns require patient to remember a preceding noun. Reduce memory demands by repeating proper noun.
Revise and restate that which was not understood.	Patients may require more than on explanation to understand concept.
<b>Use</b>	
Ask multiple choice or yes/no questions	Example: <i>"Do you want coffee or tea?"</i> or <i>"Do you want tea?"</i> <u>not</u> <i>"What do you want?"</i> This question is too open and does not provide cues for patient.
Reduce indirect speech acts.	Indirect Speech Act: <i>"Is anyone else warm?"</i> meaning I would like the temperature cooler.
Use direct speech acts.	Direct Speech Act: <i>"Please tum down the thermostat."</i>
Avoid teasing or sarcasm	Patients do not process indirect humor. Everything is taken literally.

tion in the presence of dementia progresses through three stages. The first stage deficits are primarily in the content area of lexical access and subtle conversational skills. The second stage reveals increased difficulty in content areas including concept formation, lexical access diminished graphic abilities, reliance on syntactic abilities and reduced memory function. The third stage of dementia may involve all of the above to a more severe degree, with severe memory and intellectual deficits.

Assessment batteries have been developed to evaluate the deficit areas frequently associated with communication in dementia. Clinical evaluation and standardized assessment can differentiate the language profile of dementia from other neurological disorders. Therapeutic intervention in communication focuses on maintaining the patient's communication and providing educational information for caregivers for more effective communication strategies. □

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# ALZHEIMER'S DISEASE AND CAREGIVER STRESS

LORE K. WRIGHT, PH. D., R. N. C. S.\*

Caregiving implies having responsibility for and providing the majority of direct care to an ill family member.<sup>1</sup> Approximately 80 percent of all Alzheimer's disease afflicted persons are cared for at home by family members, and caregiving responsibilities can extend over 18 years. However, the average number of years in a caregiver role is six to eight years.<sup>2</sup> It is generally acknowledged that the provision of such care is time-consuming, labor intensive, and stressful.<sup>3,8</sup> In this article, caregiver stress will be defined, the changing intensity of caregiver stress along the illness continuum will be discussed, and contributing social factors will be explored. The long-term impact of caregiver stress will be addressed as well as current efforts to reduce caregivers' stress.

## CAREGIVER STRESS

Concepts of stress, strain, or burden have been used to describe family members' experience as they provide care to an Alzheimer's disease afflicted person.<sup>7,8</sup> There is a continuing debate over how stress or burden should be defined,<sup>9,10</sup> but there is agreement that, to a great extent, burden or stress results from troublesome, disruptive behaviors associated with the insidious progression of Alzheimer's disease. In addition, many social and psychological factors contribute to caregivers' stress. In fact, the psychological aspects or the subjective interpretation of behaviors displayed by afflicted persons have been shown to be more detrimental to caregivers than actual or objective illness indicators.<sup>7,11</sup>

More recently it has been suggested that caregiving can also lead to positive experi-

ences and that negative and positive factors should be considered simultaneously when assessing stress or burden. Positive factors may result from the caregiver's sense of meeting moral obligations and the sense of reciprocating love and caring which the afflicted person had provided in the past.<sup>6</sup> There is some evidence to support of this line of thinking. Fitting et al.<sup>4</sup> found an improvement in spousal relationships after the onset of illness in 29 percent of husband and eight percent of wife caregivers. Furthermore, Kinney and Stephens<sup>12</sup> found that greater caregiver satisfaction correlated with less burden. However, high levels of stress *and* high levels of satisfaction were reported by caregivers who cared for severely impaired parents and performed a majority of caregiving tasks in conditions of financial constraints.<sup>5,6</sup>

## ILLNESS PHASES AND CAREGIVER STRESS

Variations in progression of Alzheimer's disease are well documented;<sup>13</sup> however, for the purpose of this discussion the terms early, middle, and late phases will be used to delineate different experiences of caregiver stress.

In the very early phases of the illness, a spouse or other close relative will notice changes such as increasing forgetfulness which the afflicted person tries to cover up. When important anniversary dates or other responsibilities are not remembered, a spouse may interpret such forgetfulness as neglect, rejection, relationship problems, or irresponsibility.<sup>14</sup> As one wife described her husband's early illness indications: "...I resented it, all the financial mess..., his wrecking the car and losing his job. I thought it was his fault...".<sup>15</sup> However, the afflicted person is, at least superficially, able to maintain appropriate

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social behaviors at this time, and the closest family member has trouble convincing others, including health professionals, that "something is wrong."

As the illness progresses to the middle phases and memory and functional impairments become obvious, stress or burden changes. One family member now clearly assumes a caregiver role which Mace and Rabins<sup>16</sup> so aptly describe as leading to a "36 hour day." Initial work by Zarit et al.<sup>8</sup> showed that memory problems, behavior problems, functional impairment, and duration of illness were not correlated with burden. Fitting et al.<sup>4</sup> also found that for all caregivers combined, severity of illness in a demented spouse did not predict burden. However, illness severity was significantly related to burden for younger wives and older husband caregivers.

In contrast to these earlier findings, the overwhelming majority of dementia caregiver studies show that higher levels of impairment,<sup>12,17,18</sup> increasing number of caregiving tasks and hours,<sup>5,6</sup> and especially disruptive and aggressive behaviors in the elder<sup>3,7,12,19</sup> are significantly related to higher levels of burden.

Whether demented males exhibit behaviors which are particularly stressful to female caregivers has also been investigated. Wright<sup>20</sup> found that among early to middle stage Alzheimer's disease afflicted spouses, 37 percent male but only three percent female spouses showed clinging/demanding behaviors. A few male afflicted spouses made excessive sexual demands which was particularly stressful to the respective wife caregiver.<sup>15,20</sup> Harper and Lund reported that aggressive behaviors are more severe in demented men, and such behaviors are particularly burdensome to wife caregivers. The investigators also found that daughters were more burdened when caring for a demented father than for a mother.

In the *final phase* of Alzheimer's disease, afflicted persons are less capable of expressing aggression either physically or verbally. Rather, they tend to be mute and become

totally dependent on the caregiver for all physical needs, including bathing, dressing, feeding, and toileting. To some caregivers, this illness phase is less stressful than the middle phase, unless by this time, the caregivers' own emotional and physical health has suffered as the result of caregiving.<sup>21,22</sup>

## CONTRIBUTING SOCIAL FACTORS

Cantor<sup>23</sup> observed "the closer the bond, the greater the amount of the strain." The majority of studies support that those most at risk for developing high levels of burden are spouse caregivers, wives more so than husbands, and daughters more so than sons.<sup>18,23-25</sup> However, a few studies report equal levels of burden for spouses and adult children,<sup>5,8</sup> as well as equal levels of subjective stress experienced by daughters and sons in the caregiving role.<sup>6</sup>

Whether marital status of daughters is a contributing factor is not clear. Brody et al.<sup>26</sup> reported the unmarried to be more burdened, while Miller<sup>6</sup> found married daughters to experience greater subjective stress. Married women often face competing demands between employment, caring for their own family often with young or teen-aged children, and their dependent aged parents. Brody<sup>27</sup> called them "the women in the middle" and concluded that only superwomen can deal with such role overload.

The most detailed study on kinship variations is provided by Harper and Lund<sup>19</sup> who contrast husbands, wives, and daughters subdivided according to whether the demented care recipient is at home (sharing the residence), in a nursing home, or in the community (different residence). Daughters who lived with the elder were most burdened, followed by wife caregivers with the husband at home or in a nursing home. The lowest level of burden was reported by husbands whose wives were in a nursing home.<sup>19</sup>

With respect to income some definitive observations have been made. Wife caregivers of Alzheimer's disease afflicted spouses have lower incomes than husband caregivers, and the wives have higher levels of subjective bur-

den.<sup>18</sup> Higher income lessens the burden of caregiving daughters.<sup>26</sup> In another study using multivariate analysis, income per se was not a significant predictor of subjective stress; however, care affordability (difficulties in meeting the costs for care) was a significant predictor of burden for adult child caregivers.<sup>28</sup>

Cultural and ethnic factors may also play a role in the amount of stress experienced or reported.<sup>29</sup> A number of studies show that African American and Hispanic American family caregivers of demented elders have lower perceived burden than White caregivers.<sup>30,31</sup> However, when economic status of caregivers is included in the analysis, burden is "greater among higher income than among lower income Black caregivers," but the opposite pattern is observed for White caregivers.<sup>32</sup> Stated differently, wealthier African Americans and poorer Whites have more burden while poor African Americans and wealthy Whites have less. This intriguing finding has been attributed to "disappointed aspirations" experienced by wealthier Blacks who had began to save and had dreams of a better life; but because of caregiver costs they become "nouveau poor."<sup>33</sup> Similarly, poorer Whites who probably were above the saving threshold also saw their dreams erode because of caregiving costs. For wealthy Whites on the other hand, dreamed of purchases are not likely to be affected, and for poor Blacks, disappointed aspirations are not likely to be attributed to caregiving.

### LONG-TERM IMPACT OF CAREGIVER STRESS

Given that caring for an Alzheimer's disease afflicted relative usually extends over many years, the negative impact of chronic stress on the caregivers' health is a growing concern. In an extensive literature review of health consequences of caregiver stress, Wright et al.<sup>1</sup> concluded that we have few longitudinal studies to draw definitive conclusions. What emerges from the extensive body of caregiving research, however, is a scenario of chronic stress leading to dysphoria and, in vulnerable

individuals, to depression. "Depression in caregivers may compromise the immune system which may subsequently lead to adverse physical health outcomes." The ultimate negative consequence of caregiver stress may be premature death. Preliminary findings show a death rate twice as high for caregivers than for comparable community elders over a one-year period, and female caregivers, even though younger than male caregivers, were more likely to die.<sup>34</sup>

### REDUCING CAREGIVER STRESS

Community awareness of caregivers' stress and their need for formal and informal support has grown over the past 10 years. Local Alzheimer's associations, various public agencies with demonstration grants, churches, and private organizations offer services including support groups, respite care, information, and caregiver education. These services are referred to as formal support. Informal social support by contrast consists of tangible and intangible assistance provided by family members and friends.

Research evidence of measurable positive effects of social support is conflicting. In an early study, informal support was the only variable which significantly correlated with lower burden,<sup>8</sup> and social support was particularly effective for wife caregivers whose afflicted spouses had episodes of aggressive behaviors.<sup>19,35</sup>

But not all studies show that social support (back-up help or relief time) can reduce caregiver stress.<sup>5,16,18</sup> Furthermore, women more likely than men tend to experience adverse social contacts which in turn are related to higher levels of caregiver stress.<sup>3</sup>

Group intervention programs have also been rather disappointing. While caregivers voice satisfaction with the meetings, such programs seem to have limited success in reducing caregiver stress. Toseland and Rossiter<sup>37</sup> reviewed 29 intervention studies; only two showed a reduction in burden<sup>38,39</sup> but in one of these,<sup>39</sup> a similar reduction was also experienced by the control group. A recent meta-analysis of con-

trolled studies that attempted reduce caregiver distress shows that, cumulatively, individual psychosocial interventions and respite programs are moderately effective while psychosocial interventions with groups are less so.<sup>40</sup>

Probably the most important finding regarding social support is that the caregivers' perception of quality and adequacy rather than the actual amount received is a significant factor in reducing stress.<sup>35</sup> In addition, the caregivers' active coping strategies and problem solving ability can help to reduce caregiver stress.<sup>3,36</sup>

## CONCLUSION

Major conclusions regarding caregiver stress are as follows: The insidious progression of Alzheimer's disease, and especially disruptive behaviors associated with the middle phases of the illness, lead to chronic stress in caregivers. The subjective interpretation of disruptive behaviors more so than actual events determine levels of stress. Contributing factors are co-residency with the afflicted and financial strains. Caregiver wives tend to be most burdened, followed by husband caregivers, then daughters, then sons. The chronic stress experienced by caregivers may lead to depression, an impaired immune system, subsequent ill physical health, and perhaps even premature death. Thus, even though the Alzheimer's disease afflicted person is the identified patient, attention must extend to family caregivers: individualized social support must be arranged, effective coping strategies be taught, and the caregivers health must be monitored.<sup>41</sup> □

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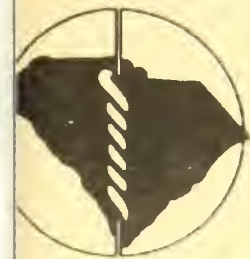


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# Continuing Medical Education

Fourth Quarter  
1994  
Calendar

James L. Haynes, M. D., Chairman

Published by the SCMA Committee on Continuing Medical Education  
Post Office Box 11188, Columbia, SC 29211

Note: CME activities in neighboring states are listed when space permits.

## OCTOBER

**Friday-Saturday**                      **Sept. 30-Oct. 1, 1994**

**Charleston, SC, The Omni Hotel**

### *War on Cancer 1994*

**SPONSOR:** Medical University of South Carolina  
**DESCRIPTION:** The objective of the conference is to update physicians on the latest cancer prevention, early detection and treatment issues.

**PROGRAM FEE:** \$250

**FACULTY:** In-state and national faculty

**CONTACT:** Odessa Ussery, (803) 792-4071

**CME CREDITS:** 8 AAFP Prescribed Hours

**Saturday-Sunday**                      **October 15-16, 1994**

**Seabrook Island, SC, Seabrook Island Conference Center**

### *Primary Care Update on Diagnosis, Management Treatment and Vascular Diseases*

**SPONSOR:** Roper Hospital

**CONTACT:** Lisa Eskridge, (803) 724-2964

**CME CREDITS:** 9.5 Hours, AMA Category 1

**Wednesday**                              **October 19, 1994**

**All hospitals and health care institutions receiving Health Communications Network broadcasts.**

### *What's New in Chemotherapy & Management of CA - A Videoconference*

**SPONSOR:** Medical University of South Carolina

**DESCRIPTION:** A live videoconference originating from the Medical University of SC with presenters from the College of Medicine and offering an opportunity for state physicians to call in their questions.

tunity for state physicians to call in their questions.

**TYPE OF AUDIENCE:** South Carolina physicians

**CONTACT:** Odessa Ussery, (803) 792-4071

**PROGRAM FEE:** None

**FACULTY:** Dr. Kapil N. Bhalla, Associate Professor of Medicine; Dr. Arthur E. Frankel, Associate Professor of Medicine

**CME CREDITS:** 1 Hour, AMA Category 1

**Thursday-Friday**

**October 20-21, 1994**

**Augusta, GA**

### *Advanced Trauma Life Support*

**SPONSOR:** School of Medicine, Medical College of Georgia

**CONTACT:** Katrinka Akesson, 800-221-6437, or (706) 721-3967

**CME CREDIT:** 17 Hours, AMA Category 1

**Thursday-Saturday**

**October 20-22, 1994**

**Myrtle Beach, SC, The Kingston Plantation**

### *Women's Health Issues*

**SPONSOR:** The National Procedures Institute

**CONTACT:** Linda Hallmann, (517) 631-4664

**CME CREDITS:** 15 AAFP Prescribed Hours

**Friday-Sunday**

**October 21-23, 1994**

**Hiawassee, GA**

### *Autumn Primary Care*

**SPONSOR:** School of Medicine, Medical College of Georgia

**CONTACT:** Katrinka Akesson, 800-221-6437, or (706) 721-3967

**CME CREDIT:** 12 Hours, AMA Category 1



*Annual Educational Symposium of the North Carolina and South Carolina Medical Directors Association*

**DESCRIPTION:** The program will feature management of peptic ulcer disease in the long-term care setting, legal liability issues for physicians practicing in the long-term care setting, managing dementia related behavior problems, update on CPT coding, use of physical restraints, and management of tuberculosis.

**TYPE OF AUDIENCE:** Physicians who practice in long-term care settings

CONTACT: Brad Whitney, (803) 457-3838;  
Margaret A. Noel, M. D., (704) 274-6182

**Columbia, SC, Dana C. Mitchell Auditorium at  
Richland Memorial Hospital**

## Founders Day

SPONSOR: USC School of Medicine

DESCRIPTION: Honoring Dr. O'Neal Humphries,  
Retired Dean for USC School of Medicine

TYPE OF AUDIENCE: Faculty, alumni, students, others

**CONTACT: Dr. Martyn Hotvedt, (804) 434-4206, ext. 4211**

**PROGRAM FEE: \$25 others**

**CME CREDITS: 3 Hours, AMA Category 1**

## NOVEMBER

**Charlotte, NC, Sheraton Imperial Hotel,  
Research Triangle Park**

***"A New Beginning...Old Problems, New Strategies"***  
SPONSOR: Minority Affairs Committee, North Carolina Academy of Physician Assistants

**DESCRIPTION:** The symposium will examine the clinical and social aspects of chronic illnesses in the minority populations and the uniqueness thereof by educating health care professionals about the available community resources which support

patients in their treatment plan.

**TYPE OF AUDIENCE:** Physician assistants, nurses, physicians, social workers, allied health professionals, and the generic public

**CONTACT:** Sonya Coble, (919) 250-8547

**CME CREDITS:** 10 Hours, AMA Category 1;  
10 Hours, Category 1 AAPA; 1.0 CEUs, Wake  
Area Health Education Center's Office

Thursday-Saturday November 10-12, 1994

## Hilton Head Island, SC, Hyatt Regency

## SCAFP 46th Annual Scientific Assembly

SPONSOR: SCAFP

**CONTACT:** Paquita P. Turner, (803) 984-7237

**CME CREDITS: 17.25 AAFP Prescribed Hours**

**Wednesday**                      **November 16, 1994**

**All hospitals and health care institutions receiving Health Communications Network broadcasts.**

## What's New in Antibiotics

**SPONSOR:** Medical University of South Carolina  
**DESCRIPTION:** A live videoconference originating from the Medical University of SC with presenters

from the College of Medicine and offering an opportunity for state physicians to call in their questions.

**TYPE OF AUDIENCE:** South Carolina physicians

**CONTACT:** Odessa Ussery, (803) 792-4071

**PROGRAM FEE:** None

**FACULTY:** Dr. J. Robert Cantey, Professor of Medicine, Dr. Bruce S. Ribner, Associate Professor of Medicine

**CME CREDITS: 1 Hour, AMA Category 1**

**Friday November 18, 1994**  
**Charleston, SC, Sheraton Inn**

## *Dying in America: Choices at the End of Life*

**SPONSOR:** Medical University of South Carolina

**DESCRIPTION:** Four of the leading medical ethicists in the country will address the topic of physician assisted suicide and the many complex and troubling issues it raises. They will help the audience clarify their own feelings and try to give insight as to how one can reach an appropriate, responsible and ethically justifiable decision.

**TYPE OF AUDIENCE:** Physicians, nurses, all



members of the healthcare treatment team, clergy, counselors, ethicists, hospital social workers and students

CONTACT: Odessa Ussery, (803) 792-4071

PROGRAM FEE: \$125 before October 17;  
\$160 after

FACULTY: Guest faculty and MUSC faculty

CME CREDITS: 6 Hours, AMA Category 1

## DECEMBER

---

**Thursday-Saturday**                      **December 1-31, 1994**  
**Charleston, SC, Department of Family Practice**  
**Naval Hospital**

### *Family Practice Lecture Series*

SPONSOR: SCAFP

CONTACT: W. Donaldson, M. D., (803) 743-7085

CME CREDITS: 8 AAFP Prescribed Hours,  
2 Elective

**Friday-Sunday**                      **December 2-4, 1994**  
**Charleston, SC, Mills House Hotel**

### *Perspectives in Pain Management*

SPONSOR: Medical University of South Carolina

DESCRIPTION: This course is designed for anesthesiologists and pain management physicians, allied health care professionals, and referring physicians from the specialties of family practice, neurosurgery, orthopaedics and pediatrics, who deal with acute or chronic pain management issues.

TYPE OF AUDIENCE: Anesthesiologists, pain management physicians and other physicians

CONTACT: Barbara Baylor, (803) 792-1607

PROGRAM FEE: \$335 Physicians/Ph.D.;  
\$230 Resident

FACULTY: Guest faculty and MUSC faculty

CME CREDITS: 14 Hours, AMA Category 1

**Monday-Sunday**

**December 5-11, 1994**

**Hilton Head, SC**

### *Psychology of Health Immunity and Disease Conference*

SPONSOR: The National Institute for the Clinical Application of Behavioral Medicine

DESCRIPTION: This conference deals directly with a broad array of topics in the behavioral medicine field. Emphasis will be upon hands-on, practicing oriented techniques for mind/body counseling and behavioral medicine.

CONTACT: Celeste N. Griffin, (203) 456-1153

FACULTY: Stephanie Simonton, Larry LeShan, Candice Pert, Leonard Laskow, and Christiane Northrup.

CME CREDITS: 20+ CEU

**Wednesday**

**December 14, 1994**

**All hospitals and health care institutions receiving Health Communications Network broadcasts.**

### *Asthma*

SPONSOR: Medical University of South Carolina

DESCRIPTION: A live videoconference originating from the Medical University of SC with presenters from the College of Medicine and offering an opportunity for state physicians to call in their questions.

TYPE OF AUDIENCE: South Carolina physicians

CONTACT: Odessa Ussery, (803) 792-4071

PROGRAM FEE: None

FACULTY: Dr. Stephen A. Sahn, Professor of Medicine, Dr. Marc A. Judson, Assistant Professor of Medicine

CME CREDITS: 1 Hour, AMA Category 1

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**Action:** Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalamic centers and release of posterior pituitary hormone.

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**Indications:** Yocon<sup>®</sup> is indicated as a sympatholytic and mydriatic. It may have activity as an aphrodisiac.

**Contraindications:** Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

**Warning:** Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

**Adverse Reactions:** Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.<sup>1,2</sup> Also dizziness, headache, skin flushing reported when used orally.<sup>1,3</sup>

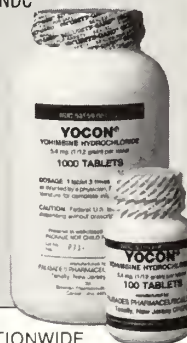
**Dosage and Administration:** Experimental dosage reported in treatment of erectile impotence.<sup>1,3,4</sup> 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to 1/2 tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.<sup>3</sup>

**How Supplied:** Oral tablets of Yocon<sup>®</sup> 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

### References:

1. A. Morales et al., New England Journal of Medicine: 1221, November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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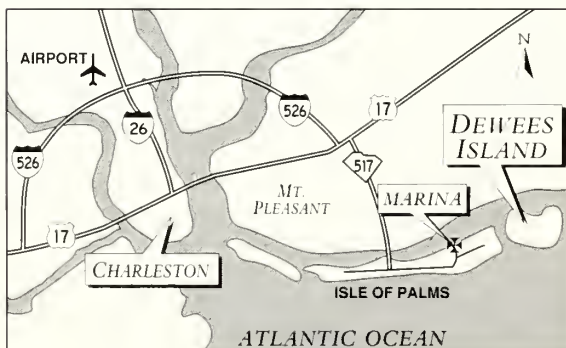
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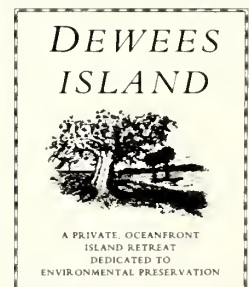
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# On the Cover:

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## ALOIS ALZHEIMER, 1864-1915

Alois Alzheimer was born in Bavaria, not far from Bamberg, which lent its name to our own South Carolina city and county. Alzheimer lived during the glory years of the Second German Reich, in a time of unprecedented scientific progress which preceded World War I. From 1882 to 1887, he attended the medical schools of the Universities of Würzburg, Tübingen, and Berlin. His life's work began at Frankfurt-am-Main, where he met the anonymous patient whose brain pathology and clinical course he so vividly described, after he had moved on to Munich. Alzheimer's name is today a household word because his patron Emil Kraepelin insisted that the newly discovered disease of the cerebral cortex be named for the physician rather than for the patient.

Alzheimer exemplified the German maxim: *Technik ist alles!* He often worked far into the night in his Munich laboratory, teaching neuropathology to students from all over the world. He received many honors during his lifetime, tragically shortened by endocarditis, which he developed in the same year he was appointed to the Chair of Psychiatry at the University of Breslau. With his colleague Franz Nissl, also a native son of Bavaria, Alois Alzheimer should be remembered as a Founder of Neuropathology second only to Rudolf Virchow, who called Pathology "The German Science."

Charles N. Still, M. D.  
W. J. B. Dorn Veterans Hospital  
Columbia, SC 29201



# Alliance Page

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## SCMAA HEALTH PROMOTION

In 1991, the AMA reported that more than four million women, one out of every three, would be assaulted by a domestic partner in any given year. Yet it has taken the murder of Nicole Brown Simpson to give national resonance to those numbers.

This year the South Carolina Medical Association Alliance will focus on the epidemic of domestic violence by not only helping victims, but also by initiating efforts to develop alternative responses to frustrations, anger and low self-esteem. The 1993 "Free to be Me" program, sticker distribution to children and adolescents, emphasized the freedom to children to choose to be drug-free and encouraged self-worth and self-respect. The stickers, along with the new AMA Alliance "I Can Choose" conflict resolution workbook, will be distributed in the local schools to encourage children to make healthy choices.

There are many other programs of importance that will be emphasized this year. This is the first year that Medical Alliance Month (March, 1995) will be observed. The goal is to promote how each Auxiliary/Alliance responds to the needs found in its community. Statewide, we will sponsor a "Walk for the Child." Through the walk, we want people to become aware of the plight of our children, especially the ones who live in poverty, the abused, and the neglected. It is our continuing emphasis on the child welfare and advocacy program, "I Have a Voice."

Governor Carroll Campbell has set a goal of having 90 percent of South Carolina's children immunized by May, 1995. To support this goal, we would like each Alliance/Auxiliary to initiate a program in its community to educate mothers about the importance of immunizing their children.

Creating smoke-free schools is another area of interest. Currently, only 29 school districts have smoke-free policies for students, faculty and staff. Change will be the result of School Board by School Board initiatives.

Other programs will include the national "Red Ribbon Campaign" to be held in the fall, the "Grow with Books," a project in conjunction with the S. C. State Library, and our most visible health promotion program, The Health Education Van.

As physicians' spouses, one of our most important roles is to educate the public on health issues and to promote safe and healthy lifestyles. Our success will be measured by the people whose lives we touch.

Mrs. Earl Curry Alford (MarKay)  
Chairman, Health Promotion



# Gray Matter

*“Matters of Interest  
to South Carolina  
Physicians.”*

Thornton & Thorne give the medical community something to think about this month.

## **URGENT MESSAGE TO FEMALE PHYSICIANS**

### **ELIMINATION OF UNISEX RATES WILL INCREASE YOUR DISABILITY INSURANCE PREMIUMS**

You are going to pay sharply increased premiums for disability insurance unless you act quickly. Companies are changing from *unisex* rates to *gender based* rates. Gender based rates mean higher premiums for females.

The disability insurance marketplace is dominated by only a few companies. Once the major companies introduce a significant change, the others are forced to follow.

Provident Life & Accident, Northwestern Mutual, Paul Revere, UNUM, and Guardian are now using sex distinct rates. These companies are market leaders so it's just a matter of time until all major companies use sex distinct

rates. Expect all companies to make the change by the end of 1994.

### **How significant is the increase?**

While it varies from company to company, one major company charges a 45 year old female physician \$3,600 per year compared to \$2,600 for her male counterpart for a policy with a \$5,000 monthly benefit, 90 day elimination period and age 65 benefit period.

### **CAN YOU PROTECT YOURSELF AGAINST THIS PRICE INCREASE?**

### **Yes, provided you act quickly!**

Premiums for noncancelable policies cannot be increased prior to age 65. Therefore if you buy your policy before a company implements this pricing change, you will save thousands of dollars over the life of the policy.

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SCMA members also receive a 25% premium discount on policies issued by Connecticut Mutual. This provides you

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**Views expressed herein are those of the authors and in no way represent SCMA.**

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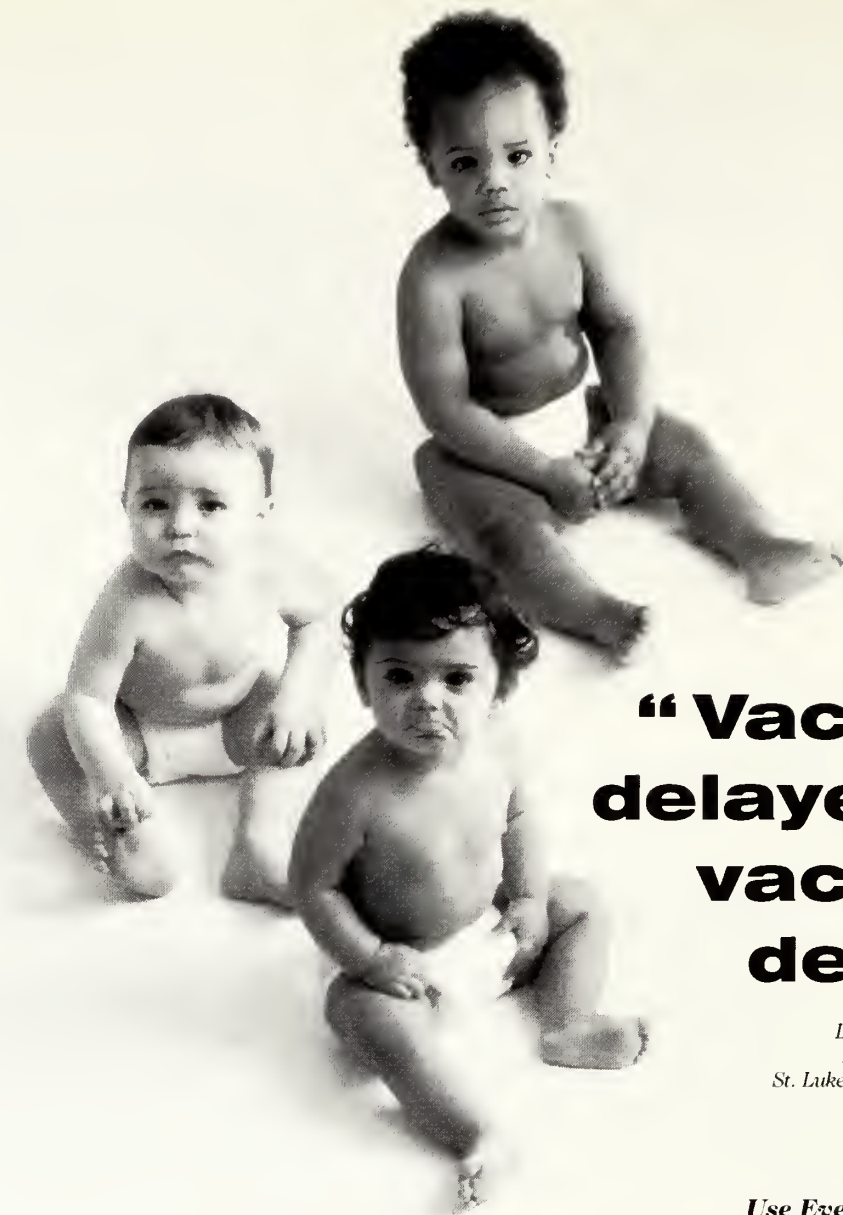
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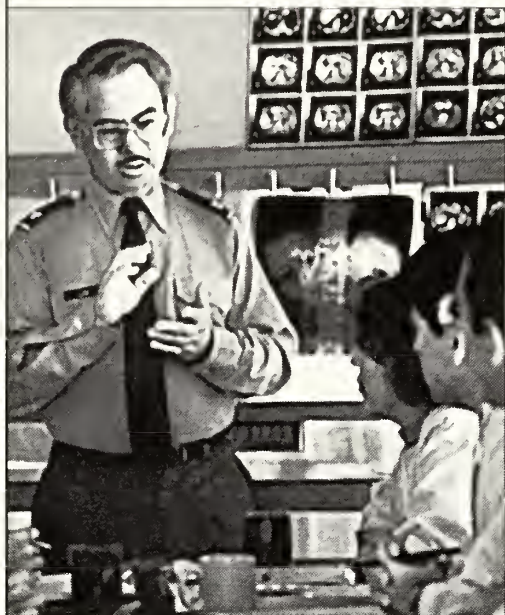
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### INDEX TO ADVERTISERS

B & B X-Ray .....	394
Anne M. Bowen, C. F. P. ....	433
The Cliffs at Glassy .....	411
Companion Technologies .....	393
CompuSystems .....	Cover 4
Deweese Island.....	434
Harry A. Hancock, LL. M. ....	Cover 3
Lexus of Charleston.....	Cover 3
The Medical Protective Company .....	412
Nationsbank .....	Cover 2
NICABM .....	433
Palisades Pharmaceuticals .....	433
SCIMER .....	439
U. S. Air Force.....	428
U. S. Air Force Reserve.....	398
U. S. Army Reserve.....	442
U. S. Naval Reserve.....	410
Winchester Surgical Supply Company .....	406



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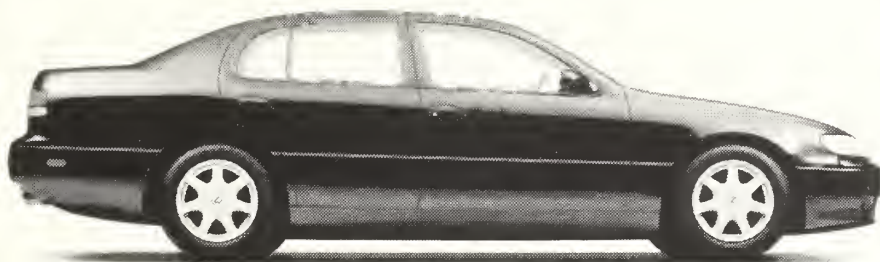
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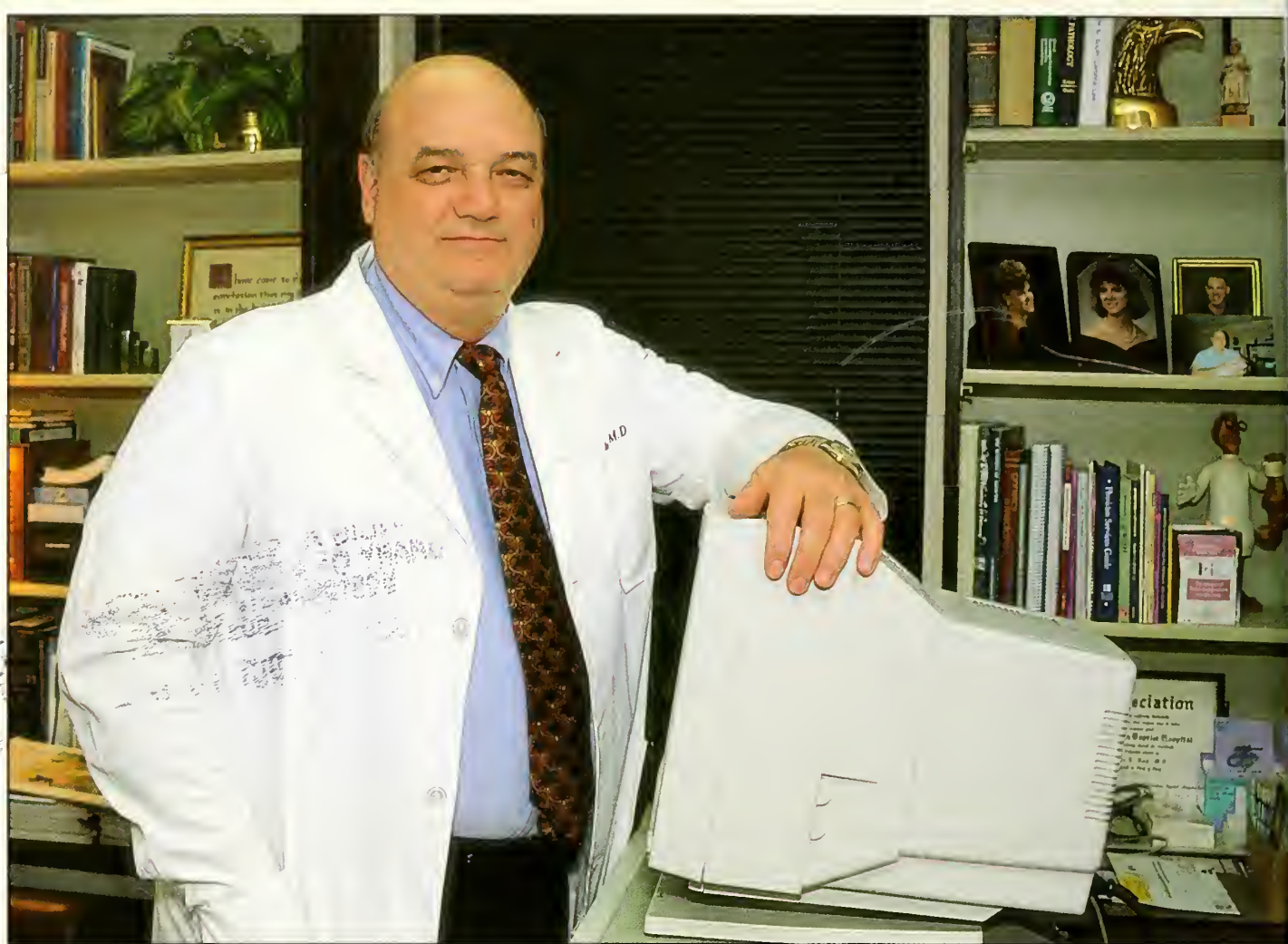
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# The Journal



OF THE SOUTH CAROLINA MEDICAL ASSOCIATION

VOLUME 90  
NUMBER 10  
OCTOBER 1994  
PAGES 443-528

**SPECIAL ISSUE: UPDATE ON OTOLARYNGOLOGY -  
HEAD AND NECK SURGERY**

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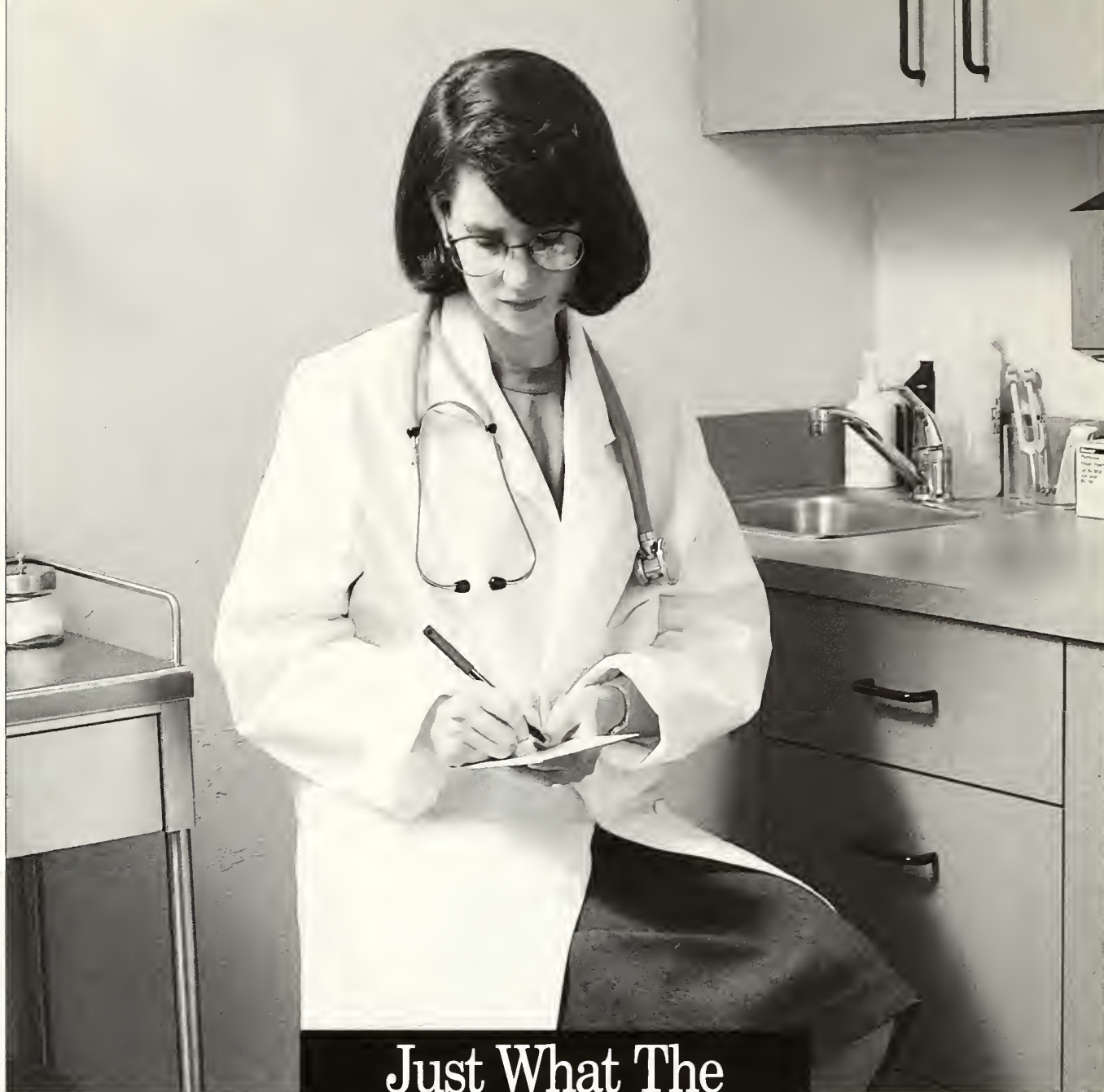
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# President's Page

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## STAY WITH THE PACK

Dr. Lonnie Bristow, President-elect of the American Medical Association, has on several occasions emphasized to physician audiences that "the strength of the wolf is in the pack, and the strength of the pack is in the wolf." His obvious analogy is that organized medicine is the pack and the individual doctor the wolf. The message is that alone, individual practitioners have little power, but as a part of the House of Medicine have significant clout. Conversely, the AMA (or the SCMA) cannot succeed without strong individual members. Dr. Bristow's challenge for us is to stay together during health system reform so that the profession of medicine and patients will be the winners. The physician cannot prevail without the AMA/SCMA and the AMA/SCMA cannot be victorious without the physician. If this national leader in medicine is correct, and I am firmly convinced he is, it is a classic win-win or lose-lose scenario for both parties. The right decision for each of us is to stay together on *major* issues that affect our chosen field despite minor differences between individuals and specialties.

Our Palmetto state is about to undergo an unprecedented transformation of its health care marketplace similar to the tumultuous and often disruptive events that have already affected providers and patients elsewhere (California, Texas, etc.). Many of the power, control, money and clinical autonomy issues will begin as struggles between providers and large health care companies, but the final decisions are going to be made by the courts and the South Carolina Senate and House of Representatives. You can be certain that the opposing side (insurance companies, health care corporations, chambers of commerce, etc.) will hang together in these conflicts. In fact they have already begun to exhibit this behavior in their strong and organized opposition to the AMA's Patient Protection Act and antitrust proposals.

I have proposed and have obtained Board of Trustees approval to establish an interspecialty council which will have members from each medical and surgical discipline represented in the SCMA House of Delegates. As of this writing, 15 specialty societies have already appointed their representative. Meetings will begin biweekly during the 1995 legislative session. Pertinent bills and proposals will be monitored as they proceed through committee actions and consensus opinion involving each specialty will be sought. SCMA legislative staff will use the advice of this council in its day-to-day interactions with members of the general assembly and in testimony when appropriate. It is important that each one of us identify our specialty representative and communicate our individual thoughts regarding specific issues. We need to support consensus opinion and compromise. It is critical that every SCMA member physician support this initiative and other efforts toward professional unity as we enter the troubled waters which lie ahead. Finally, do not be a lone (and weak) wolf. Stay with the pack.

*O. Marion Burton MD*  
O. Marion Burton, M. D.  
President

## Contents

### *Special Issue: Update on Otolaryngology - Head and Neck Surgery*

*Guest Editors: M. Hochman, M. D.  
J. David Osguthorpe, M. D.*

**See page 447 for complete list of contents**

### *Editorial*

#### **523 Editorial**

*Marcelo Hochman, M. D.*

### *Features*

- 524 Alliance Page**
- 523 On the Cover**
- 445 President's Page**

### *Association*

- 511 Gray Matter**
- 515 Physician Recognition Award**
- 475 SCMA Newsletter**

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## SPECIAL ISSUE: UPDATE ON OTOLARYNGOLOGY – HEAD AND NECK SURGERY

GUEST EDITORS: M. HOCHMAN, M. D.  
J. DAVID OSGUTHORPE, M. D.

- |   |   |
|---|---|
| <p><b>448 Introduction</b><br/><i>M. Hochman, M. D., David Osguthorpe, M. D.</i></p> <p><b>450 Multi-disciplinary Approach to Cranial Base Tumors</b><br/><i>J. David Osguthorpe, M. D., Sunil Patel, M. D., Marcelo Hochman, M. D., Gene Howard, M. D., Joseph Jenrette, M. D., James Cooke, M. D., Joseph Horton, M. D., Peter Weber, M. D.</i></p> <p><b>455 The Management of Non-melanoma Skin Cancer of the Head and Neck</b><br/><i>Pearon G. Lang, M. D., Marcelo Hochman, M. D.</i></p> <p><b>479 Comprehensive Care of the Cleft Lip and Palate Patient</b><br/><i>William C. Giles, M. D., Paul T. Davis, M. D.</i></p> <p><b>483 Otitis Media: When are Tubes and/or Adenoidectomy Needed?</b><br/><i>Jacquelyn A. Going, M. D., Daniel M. Ervin, M. D.</i></p> <p><b>485 Hearing Aids: Selection, Parameters, &amp; Limitations</b><br/><i>Robert M. Poland, M. A., M. A., CCC-A, Robert C. Waters, M. D., Julie A. Newburg, M. D.</i></p> <p><b>491 Cochlear Implants</b><br/><i>Timothy Courville, M. D.</i></p> | <p><b>495 Snoring and Sleep Apnea: Evaluation and Treatment</b><br/><i>John G. Phillips, M. D., Charles E. Smith, M. D., D. M. D.</i></p> <p><b>499 Hay Fever: Pharmacotherapy or Immunotherapy?</b><br/><i>William J. Fravel, M. D., Theodore A. Watson, M. D.</i></p> <p><b>501 If Surgery is the Last Option for Sinusitis, what is the First?</b><br/><i>Gregory S. Parsons, M. D., Brian C. Wilson, M. D.</i></p> <p><b>505 Common Voice Disorders</b><br/><i>Jane W. Riester, M. D., Michael Hellstrom, M. D.</i></p> <p><b>507 Options in Cosmetic Rehabilitation of the Aging Face</b><br/><i>Thomas Fincik, M. D., James F. White, M. D.</i></p> <p><b>513 Evaluation of the Neck Mass</b><br/><i>Jeffrey R. Fenwick, M. D., Russell D. Kitch, M. D.</i></p> <p><b>517 The Evaluation and Diagnosis of the Dizzy Patient</b><br/><i>W. David Isenhowe, Jr., M. D., Richard M. Carter, M. D., Peter C. Weber, M. D.</i></p> |
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# OTOLARYNGOLOGY – HEAD AND NECK SURGERY UPDATE: INTRODUCTION

GUEST EDITORS: M. HOCHMAN, M. D.\*  
DAVID OSGUTHORPE, M. D.\*

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Members of the South Carolina Society of Otolaryngology-Head and Neck Surgery have assembled the third, in a five-year cycle, update for their primary care colleagues. Nine of the articles address common questions in physician referrals to our specialty. Drs. Going and Ervin of Georgetown and Myrtle Beach, respectively, present an interim “best guess” on the management of otitis media, a disease which affects at least one-third of children. The selection and duration of antibiotic therapy, and the parameters for pressure equalization tubes, are questions most of us address on a weekly basis. Almost as common as otitis media, sinusitis affects most adults at some time. A preponderance of these infections respond to pharmacotherapy, and a conservative treatment regimen is detailed by Drs. Parsons and Wilson of Rock Hill. Inhalant sensitivities are primary contributors to chronic sinusitis, and Drs. Fravel and Watson of Columbia and Greenville, respectively, detail anti-allergy pharmacotherapy and reasons for immunotherapy. Nasal obstruction is one of many potential causes of snoring and sleep apnea, which include redundant soft palate and pharyngeal tissues, base of tongue prominence, mandibular hypoplasia, parapharyngeal fat deposition, central neurologic disorders and the like. An approach to the evaluation and management for snoring is presented by Drs. Smith and Phillips of Greenville. Whether chronic nasal congestion with post-nasal drip, acid reflux, vocal abuse or a neurologic disorder, voice disorders affect many. A

straightforward approach to the diagnosis and conservative management of hoarseness is presented by Drs. Riester and Helstrom of Anderson. As with hoarseness, cancer is likely both a patient’s and their physician’s greatest concern with a lump in the neck, so Drs. Fenwick and Kitch of Summerville present a protocol for evaluating a neck mass. As the population ages and the noise exposure in our “modern” lifestyle takes its toll, hearing loss is prevalent. Auditory evaluation and the decision parameters salient to hearing aid selection must not be deferred to hearing aid dealers. Drs. Waters, Newburg and Poland of Easley detail the pros and cons of the various styles of hearing aids, and the limitations of aural rehabilitation. Prevalent in the same age group are balance disorders, globally termed “dizziness” by most patients. Drs. Isenhower and Carter of Greenwood present the differential diagnosis and simple, straightforward evaluation which will spare the majority of referral for more expensive tests such as magnetic resonance imaging and electronystagmography. Cosmetic rehabilitation of the aging face is a frequent topic in the lay press, and generates many questions to physicians regarding what is feasible and realistic. Drs. Funcik and White of the Isle of Palms and Columbia, respectively, detail common techniques for facial rejuvenation.

Multi-disciplinary approaches to difficult clinical problems pool the strengths of various specialties and are usually much to the patient’s benefit. Collaboration in the management of the cleft palate patient has occurred for at least two decades, where the high profile surgical repair must be augmented by attention to eustachian tube function and otitis

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media, by fostering speech development and by the correction of malocclusion, as outlined by Drs. Giles and Davis of Columbia and Florence, respectively. The article by Drs. Hochman and Lang of Charleston outlines the treatment options for skin neoplasms, the most common cancer affecting the human population. The skills of dermatologist, facial plastic surgeon and radiation therapist may be required for successful management. Even wider inter-specialty collaboration is required for skull base tumors, with the skills of a neu-

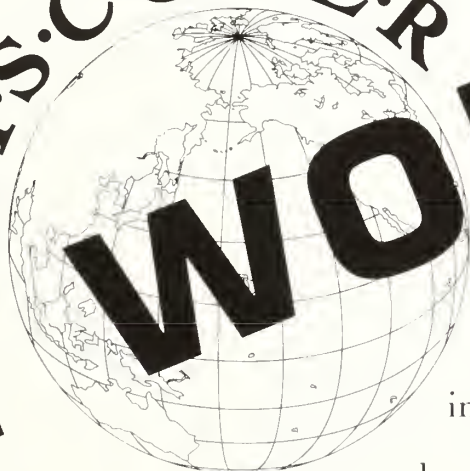
rosurgeon, head and neck cancer surgeon, oculoplastic surgeon, neuroradiologist and radiation therapist frequently involved in this state-of-the-art pursuit.

The South Carolina Society of Otolaryngology-Head and Neck Surgery, the authors of the papers in this issue of *The Journal* and the guest editors wish to thank Proctor and Gamble Pharmaceuticals, Inc., for the generous donation which supported the increased number of articles in this issue. □



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# MULTI-DISCIPLINARY APPROACH TO CRANIAL BASE TUMORS\*

J. DAVID OSGUTHORPE, M. D.\*\*

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## INTRODUCTION

Tumors involving the skull base pose substantial challenges to effective therapy. For most of this century, such lesions were usually "debulked" by a neurosurgeon or otolaryngologist, utilizing intracranial or extracranial approaches, respectively, and then subjected to palliative irradiation and/or chemotherapy. The first joint neurosurgical and otolaryngologic team approaches to cranial base tumors were reported in the 1950s, and there has since been a gradual proliferation of multidisciplinary teams in Europe, Japan and the United States, though still less than 15 in the latter. With a team approach and improvements in technology, cure rates for cranial base neoplasms, near zero in the first half of this century, now exceed 50 percent for benign tumors, and as high as 80 percent for meningiomas and angiofibromas, with more modest 20-30 percent cure rates for malignancies of the temporal bone, paranasal sinuses or nasopharynx

which have penetrated the cranium. Common components for the cranial base team include: (1) a neurosurgeon with oncology and craniofacial subspecialization, (2) a head and neck cancer surgeon with interest in the sinuses, orbits and ear, (3) a facial plastic and reconstructive surgeon with free flap transfer expertise, (4) an oculoplastic surgeon, (5) a neuro-radiologist with embolization skills, (6) a radiation oncologist with radiosurgery training, (7) an anesthesiologist with experience in hypotensive anesthesia and nerve/EEG monitoring, and (8) a support team of prosthodontists, vocational rehabilitation nurses, social workers, speech and swallowing therapists, audiologists and the like.

## DISCUSSION

The neural and vascular involvements of a cranial base tumor commonly determines resectability and, if resection is possible, most of the functional sequelae of extirpation. For these reasons, the intracranial portion of a "team" resection is performed first, unless extracranial control of the internal carotid artery or internal jugular vein are required.<sup>1</sup> Cutaneous incisions are frequently disguised, such as behind the hairline for an extended frontal (coronal) approach to anterior fossa, orbital and sinus neoplasms. To minimize brain injury from the prolonged retraction in these procedures, the craniotomies are placed

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\*From the Departments of Otolaryngology and Communicative Sciences (Drs. Osguthorpe and Hochman), Neurosurgery (Dr. Patel), Ophthalmology (Dr. Howard), Radiation Oncology (Dr. Jenrette), Anesthesiology (Dr. Cooke), and Radiology (Dr. Horton), Medical University of South Carolina, Charleston.

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low on the skull, so that retraction can be divided between extracranial and intracranial structures. For instance, most sinus or orbital neoplasms which have penetrated the anterior fossa are approached through a "subfrontal" craniotomy which includes only a few centimeters of the lower frontal bone, but encompasses the bridge of the nose and superior orbital rims<sup>2,3</sup> (Figures 1 and 2). Further posterior inclusion of the orbital roofs and ethmoids provides exposure for large anterior fossa meningiomas, extensive sella and sphenoid masses, and clival tumors reaching, if necessary, to the anterior lip of the foramen magnum<sup>3</sup> (Figures 3 and 4). For malignant tumors, despite magnetic resonance images, abutment versus invasion of intracranial structures must sometimes be determined intraoperatively, and a case can be aborted if intracranial inspection reveals the brain/nerve sacrifice necessary for a resection would cause excessive morbidity. For example, a unilateral sphenoid carcinoma may invade the ipsilateral cavernous sinus and internal carotid artery, which can usually be extirpated from a technical standpoint, but would require sacrifice of cranial nerves 2-6, and risk stroke.<sup>4</sup> The risks/benefits of an extirpation, and the realistic expectations of cure, are frankly discussed preoperatively with the patient and their sig-

nificant others.<sup>1,5</sup>

Utilizing craniofacial osteotomies developed for the correction of congenital midfacial deformities, the head and neck surgeon can approach any region along the skull base.<sup>1-3,5</sup> The sinuses and orbits are accessed through a lateral rhinotomy or midfacial degloving, the nasopharynx through a parotidectomy and lateral mandibular mobilization, (Figure 5) and middle and posterior fossae through the temporal bone. For the neurosurgeon, these, when added to the usual craniotomies and/or temporal bone osteotomies, provide a shorter working distance to the more midline skull base tumors such as petroclival meningiomas, vertebro-basilar aneurysms, large acoustic neuromas and cavernous sinus lesions. Aside from unilateral deafness or blindness from temporal bone or globe/optic nerve invasion, most of the extracranial accesses involve minimal to modest disruption, as the cranial and facial bones are plated back into position and nerve defects grafted.<sup>6</sup> Although orbital invasion by sinus or intracranial neoplasms may necessitate exenteration, frozen section margin control and microdissection within the orbit can allow preservation of vision in selected cases when the globe and optic nerve are uninvolved.<sup>7</sup> The orbital walls are reconstructed with bone grafts and absent extraocular mus-

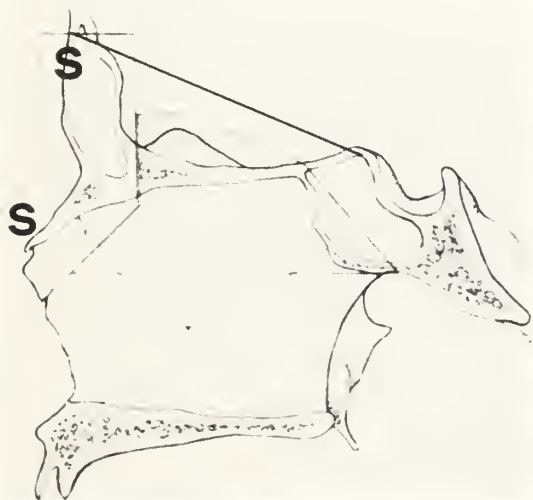


Figure 1. Schematic representation of sagittal view of a "low frontal" osteotomy exposure of the nasal vault, cribriform plate, sphenoid, sella and clivus (from Reference 2).

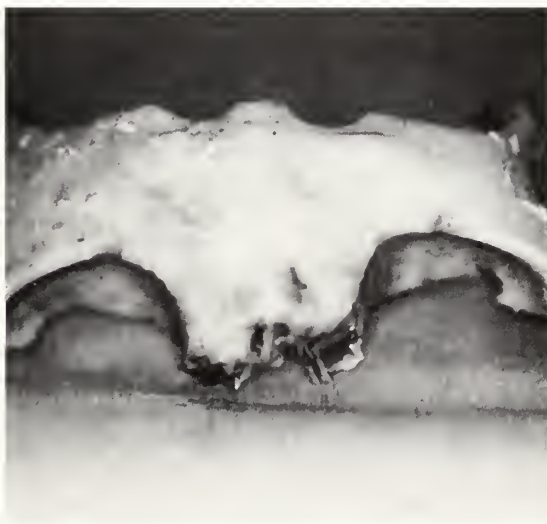


Figure 2. Note superior orbital rims, base of nasal bones and lower portion of frontal bones removed en bloc for "low frontal" access to a sphenoid and clival meningioma.



Figure 3. Computed axial tomograms of a large clival and cavernous sinus meningioma, with displacement of both carotid arteries and optic nerves. Saphenous nerve bypass graft required for one carotid.

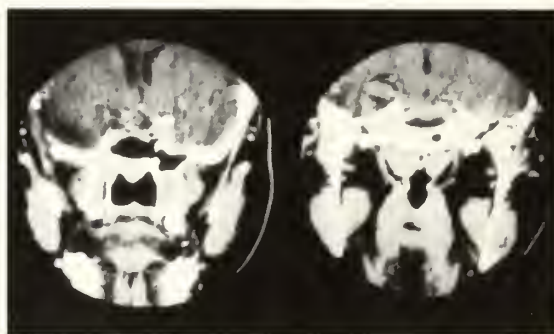


Figure 4. Post-operative computed axial tomograms of patient in Figure 3.

cle segments bridged with sutures or grafts.

In the past, patients surviving craniofacial ablations were left with substantial deformities and functional limitations. The reconstructive team must not only separate the intracranial contents and cerebrospinal fluid from the extracranial field, but also reconstruct bony defects, augment soft tissue deficiencies and otherwise correct form and function. Local scalp and/or temporalis muscle flaps have been traditional, but are being supplanted by free tissue transfers with microvascular anastomoses.<sup>4</sup> Thin soft tissue defects may be resurfaced with a radial forearm flap, and wider/thicker defects with a rectus muscle and abdominal skin flap. Though rib or outer table of cranial bone grafts suffice for most bony defects, vascularized bone transfer from the fibula, scapula or iliac crest are preferable when both bone and soft tissue augmentation are needed for a particular defect. When a prosthesis is necessary, osteointegrated metal posts allow stable fixation and spare the patient daily gluing, and the embarrassment of prosthesis slippage in social or athletic situations. Eyelid malpositions, when a facial nerve or oculomotor nerve are sacrificed, are corrected with gold weights, and static or dynamic slings.

Most cranial base surgery is accomplished under normotensive anesthesia; however, hypotension is preferred when the surgeons work in a highly vascular field. Intraoperative EEG provides feedback regarding cranial blood flow should temporary or permanent carotid occlusion be necessary, and indicates whether a bypass graft is required. Brainstem

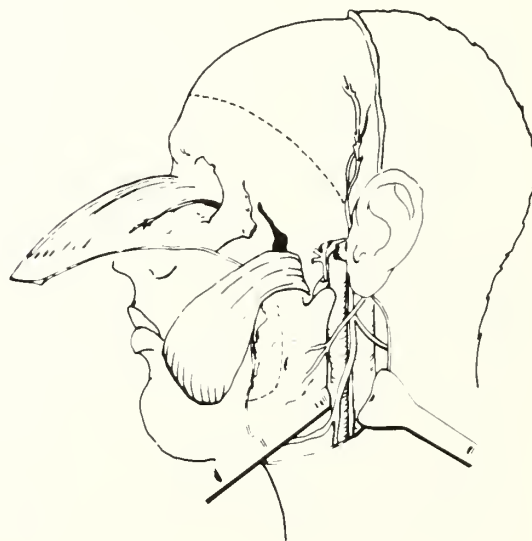


Figure 5. Schematic representation of surgical access to lateral cavernous and infratemporal fossa lesions.

auditory evoked potential and somatosensory evoked recordings are utilized when the tumor must be removed from the brainstem or when cerebral retraction is necessary. The integrity of cranial nerves can be monitored, with one probe placed intracranially proximal to the tumor, and the other probe on the distal, facial portion of a nerve or the muscle it innervates (cranial nerve EMG's). Dissection in particularly vascular tumor areas, such as the cavernous sinus, occasionally requires the cardiopulmonary bypass technique developed for aneurysm repairs whereby the patient is infused with steroids and barbiturates, cooled to 15-20° centigrade, and cardiac standstill induced. This allows up to 30 minutes of essentially bloodless dissection.

Preoperative delineation of skull base tumors is critical to determining whether a resection is technically feasible, which structures must be sacrificed (and, hence, the morbidity of a resection) and the degree of tumor vascularity. Thin slice computed tomography affords bony detailing, critical in the temporal bone, clival, parasellar and cavernous regions. Magnetic resonance imaging, enhanced by surface coils, delineates soft tissue tumor, and multi-planar reconstructions provide a 3-D image with which to plan an approach for best exposure and least morbidity. Magnetic resonance angiography may sufficiently detail vessels feeding a tumor, but arteriography is preferred when tumor embolization or carotid artery sacrifice are anticipated (Figure 6). If temporary or permanent occlusion of an internal carotid flow is likely, a preoperative trial balloon occlusion of that artery, with the patient awake to assess the neurologic consequences, is performed preoperatively. The findings are integrated with pre- and post-occlusion brain scans to delineate subtle areas of impaired perfusion and, thus, predict stroke with permanent occlusion. The extracranial portion of vascular tumors are commonly embolized preoperatively to diminish intraoperative blood loss, and in recent years improvements in techniques and catheters have allowed intracranial embolization in

many cases as well.

Most benign tumors and many low grade malignancies about the cranial base are relatively radioresistant. In addition, treating invaded bone without causing osteonecrosis, and limiting dose delivery to vital neurovascular structures, makes irradiation cure infrequent. The recent widespread availability of higher energy sources has improved the delivery of effective doses to bone, but the limited radiation tolerance of central nervous system tissue remains a problem. One promising technique is radiosurgery, which is stereotactically-focused irradiation with a high dosage gradient to spare adjacent tissues, but field size is usually limited to 3-4 cm. Long-term experience is still being accrued in Europe where the technique was introduced over a decade ago, and we have a three-year experience in South Carolina. Cures are infrequent

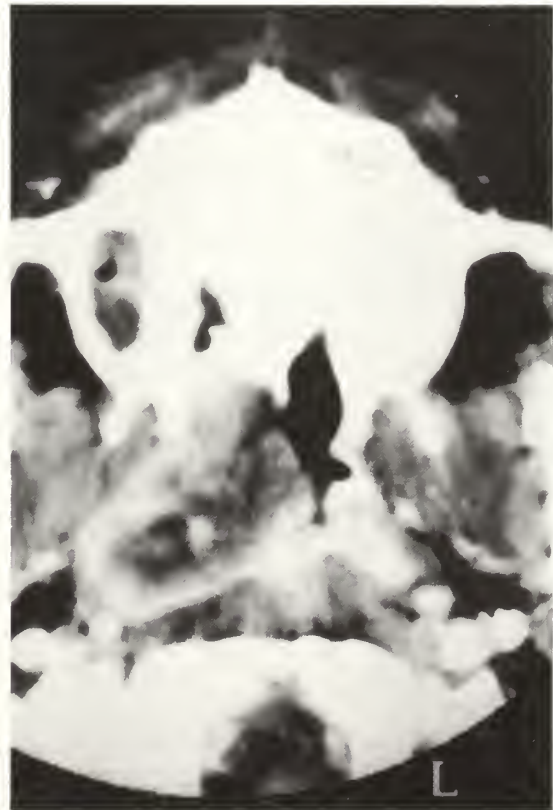


Figure 6. Computed axial tomograms from 35-year-old-male with a nasopharyngeal carcinoma which had failed to respond to irradiation and chemotherapy. Note encroachment by tumor of one carotid artery (arrow).



for benign tumors, but long periods of growth arrest are common, whereas malignancies have a rapid initial response yet poor long term prognosis. Intraoperative irradiation, still an experimental therapy, entails transferring the patient intraoperatively to a sterile irradiation facility for a single, high dose treatment to the tumor bed before wound closure.

By the time of hospital discharge after a cranial base procedure, patient and family concerns regarding operative complications have faded, and restoration of appearance and a return to activities of daily living become primary concerns. Appropriate consultation with a vocational rehabilitation counselor, speech pathologist, physical therapist and prosthodontist help to mold the cranial base team's goals of curing the patient of disease and restoring functional and social aspects of their life.

In summary, the cranial base team is yet another example of how patients benefit from a combined approach to complex problems with the end result, by summing the strengths

of multiple medical disciplines, being much better than any individual portion thereof. □

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# THE MANAGEMENT OF NON-MELANOMA SKIN CANCER OF THE HEAD AND NECK\*

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## EPIDEMIOLOGY

Skin cancer is the most common malignancy occurring in man. Basal cell carcinomas (BCC) constitute the majority of non-melanoma skin cancers (NMSC) but 20 percent are squamous cell carcinomas (SCC).<sup>1</sup> In recent years, there has been an alarming increase in the incidence of skin cancer.<sup>2,3</sup> The American Cancer Society predicts that during 1994, 700,000 skin cancers will be diagnosed in this country. Ninety percent of these will be non-melanoma in nature. The current epidemic of skin cancer probably reflects changes in dress and lifestyle which allow for greater sun exposure. However, the depletion of the ozone layer, which filters out carcinogenic ultraviolet rays, will almost certainly contribute to the development of skin cancer in future generations.<sup>4</sup>

## PATHOGENESIS

The most common factor involved in the pathogenesis of NMSC is ultraviolet light (UVL). The UVL rays most important in cutaneous carcinogenesis are in the range of 290-320 nm.<sup>5</sup> However, artificial sources of UVL, including tanning beds<sup>6</sup> and PUVA therapy for psoriasis,<sup>7</sup> may contribute to the genesis of NMSC. The typical patient who develops NMSC has a fair complexion, burns easily and tans poorly.<sup>8</sup> Often they have a history of chronic sun exposure either as a result of occupational exposure or recreational pursuits.<sup>9</sup> Men are more commonly affected than women.<sup>10</sup> Chronic sun exposure correlates

better with the distribution of SCCs than it does with BCCs. Approximately one-third of all BCCs occur in areas of relatively minimal sun exposure.<sup>11</sup>

Besides UVL, other factors may contribute to the development of NMSC. The importance of genetics is typified by patients with the basal cell nevus syndrome<sup>12</sup> and xeroderma pigmentosum.<sup>13</sup>

Trauma, such as burns and cuts, has been associated with the "acute" onset of NMSC.<sup>14</sup> Non-melanoma skin cancer may arise in scars of various etiologies including those associated with vaccinations,<sup>15</sup> burns,<sup>16</sup> and discoid lupus erythematosus (DLE).<sup>17</sup> Chronic stasis ulcers and sinuses associated with osteomyelitis also may give rise to NMSC.<sup>18</sup> SCCs are more likely to arise in an old burn scar or chronic sinus tract or ulceration than a BCC. These SCCs have the reputation of carrying a poor prognosis with a high risk for recurrence and metastases (10-30 percent). However, in reviewing the literature it is difficult to know if these SCCs are intrinsically more aggressive or if their reputation is more likely due to a delay in diagnosis and treatment. Compared to whites, a greater proportion of SCCs in blacks arise in association with pre-existing conditions such as scars, chronic ulcers and sinuses, and DLE.

Radiation exposure, either through occupational exposure, or exposure during the treatment of benign conditions such as acne, hirsutism, and tinea capitis may lead to the development of NMSC years later.<sup>21</sup> SCCs arising in areas of prior irradiation are said to possess a significant propensity for metastasis (10-30 percent).<sup>22</sup>

Immunosuppression due to lymphoproliferative diseases, AIDS, transplantation, and the

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use of immunosuppressive agents has been associated with an increased incidence of NMSC especially SCC.<sup>26,27</sup> In such patients, the tumor is more likely to behave aggressively and possesses a significant propensity to metastasize. In transplant patients the true propensity for developing multiple NMSCs may not be realized for several years after transplantation. The frequency with which a transplant patient develops cutaneous carcinomas correlates strongly with the intensity of their sun exposure.

### MODES OF SPREAD

Non-melanoma skin cancers always follow the path of least resistance. Thus these tumors, at least early on, are more likely to spread along the perichondrium, periosteum, tarsal plate or fascia rather than invade cartilage, bone, or muscle. This is especially true of BCCs.<sup>28</sup> This type of spread can result in an "iceberg phenomenon" with extensive subclinical spread and, in part, explains the high recurrence rates reported for NMSC of the nose, ear, scalp, and periocular area. On the distal nose, the tumor can spread along the perichondrium of the articulating cartilages until it comes to a soft tissue plane where it can easily penetrate the substance of the nose. Embryonic fusion planes (e.g., nasolabial fold, retroauricular sulcus, preauricular area, inner canthus, philtrum, and mid-lower lip and chin) offer little resistance to the spread of tumor and allow for extensive subclinical spread of tumor, again accounting for the high recurrence rates reported after treatment of tumors in these anatomical locations.<sup>28</sup>

Scar tissue offers resistance to tumor penetration; thus tumor buried beneath scar tissue is more likely to spread laterally or penetrate deeply, rather than try to reach the surface. Such behavior may result in a delay in diagnosing a recurrence and may allow for substantial subclinical spread. Thus, the patient may be shocked at the size of the surgical defect after the removal of a recurrent tumor. Moreover, this behavior accounts for the higher recurrence rates associated with recur-

rent tumors.

When using flaps to reconstruct defects resulting from the removal of a cutaneous neoplasm it is essential that the wound be tumor free. Tumors buried beneath flaps may not surface for years and the extensive undermining associated with the generation of the flap opens up tissue planes which offer little resistance to the subclinical spread of tumor.<sup>29</sup>

Non-melanoma skin cancer, especially SCC, may spread along vessels or nerves, especially the latter.<sup>30-32</sup> Perineural spread may be very limited, or extensive. The tumor may travel along a major nerve, enter a foramina and spread to the central nervous system.<sup>30</sup> The tumor need not be recurrent or large.<sup>32</sup> Indeed, once the clinically obvious tumor is removed, the remaining tumor may be confined solely to the perineurium.<sup>30,31</sup> Although pain, paresthesias, and paralysis are often suggested as clinical clues to perineural involvement, these findings occur late and are an ominous sign.<sup>30,31</sup> Extensive perineural spread may be present before any of these signs or symptoms develop.<sup>31</sup> Perineural involvement often will not be detected histologically if a shave excision or limited biopsy is performed. Thus if perineural involvement is suspected a deep incisional biopsy is recommended. Patients with perineural tumor spread are at high risk for recurrence.<sup>30,31,32</sup>

Metastases with BCC are rare occurring in .0028 percent<sup>33</sup> to .1 percent of patients.<sup>34</sup> The tumors which metastasize have usually been present for many years and treated on numerous occasions without success. Although any histological variant may metastasize, those with a metatypical, adenoidal or infiltrating growth pattern seem to do so more frequently. The role of prior irradiation in predisposing to metastases is controversial.<sup>33,34</sup> Why certain BCCs metastasize is not clear, however, in some patient immune impairment may be responsible.<sup>33,35</sup> Metastases most commonly occur via the lymphatics to the regional nodes or via the bloodstream to the long bones and lungs.<sup>33,34</sup>

Squamous cell carcinoma has a much



greater propensity to metastasize than does BCC. Although invasive SCCs < 1 cm in diameter may metastasize,<sup>38</sup> most are > 2 cm in size.<sup>37,38</sup> Since size often correlates with depth of invasion, it may be that depth of penetration is more important than size.

As noted earlier, SCCs arising in scars,<sup>16</sup> chronic ulcers and sinus tracts,<sup>18</sup> and areas of prior radiation<sup>22</sup> are said to have a significant incidence of metastases as well as mortality.

Squamous cell carcinomas which are recurrent are more likely to metastasize.<sup>37,38</sup> This finding stresses the importance of trying to cure the patient at the time of initial treatment.

SCCs, arising on sun protected skin, in the absence of predisposing causes are said to exhibit a significant risk for metastases (17 percent).<sup>41</sup> The explanation for this is not clear.

As noted earlier, patients with impaired immunity are more likely to develop metastases.<sup>26,27</sup>

Squamous cell carcinomas arising on actinically damaged skin or in pre-existing solar keratoses usually behave in a relatively benign manner.<sup>42</sup> Unfortunately, many clinicians have interpreted this data to mean that such SCCs do not need to be treated as aggressively as SCCs of the skin arising in other settings. Squamous cell carcinomas arising on sun-damaged skin can at times be very aggressive and should be treated with appropriate respect.<sup>37,39,43</sup> The reported incidence of metastases with these SCCs varies from .5 percent<sup>42</sup> to 10 percent.<sup>43</sup>

## MANAGEMENT OF BCC AND SCC GENERAL PRINCIPLES

In managing patients with BCCs and SCCs the physician should keep four goals in mind: (1) total removal or destruction of the tumor; (2) maximal preservation of normal tissue; (3) preservation of function, and (4) optimal cosmesis. Obviously, the first goal is most important because if this goal is not achieved, ultimately the other goals will not be achieved.<sup>57</sup>

Elderly patients should not be denied defini-

tive therapy based on age considerations alone. With people living longer and enjoying better health, with technological and surgical advancements, and with better pre-operative preparation and post-operative support systems, these patients should be offered the same treatment options as younger individuals.

Prevention is as important as treatment in patients predisposed to NMSC. Such patients should use a sunscreen with a sun protective factor > 15 on a daily basis and be judicious in exposing themselves to the sun. Sunscreens have both in animals and man been shown to decrease the risk of developing a skin cancer.<sup>58,59</sup> The frequency of follow-up visits in patients with NMSC is based upon how much trouble they have had in the past and how great the risk they will develop recurrent disease. The frequency of such visits can vary from every three months to once a year. Although some actinic keratoses will spontaneously regress and only a few progress to SCC,<sup>60</sup> since it is impossible to know which lesion will undergo this evolution or when, most physicians choose to treat these precancerous lesions.<sup>61</sup>

Topical 5-FU and masoprocol have been shown to be effective in the management of solar keratoses<sup>62</sup> and it has been implied that topical 5-FU may decrease the frequency of new skin cancers.<sup>59</sup> The role of topical tretinoin in the management of actinic keratoses and the prevention of skin cancer remains to be shown.<sup>63</sup> Although capable of reversing photodamage, the effect of tretinoin is not permanent and requires maintenance therapy.<sup>64,65</sup> Topical tretinoin is less effective than 5-FU in the management of solar keratoses.

In managing surgical defects, if a flap is used to repair the defect it is mandatory that the surgeon be sure there is no residual tumor for the reasons outlined under the section on "Modes of Spread." Burying tumor can have disastrous consequences.

If there is any question regarding the completeness of tumor removal, the defect should be allowed to heal by second intention or

covered with a split thickness graft or the patient fitted with a prosthesis (where appropriate). The patient can then be followed until it appears that it is safe to carry out definitive reconstruction.<sup>29</sup> In patients at high risk for recurrence, we prefer to follow them for at least a year before carrying out their reconstruction.

In addition, in patients with extensive sun damage, the donor site for a flap should be closely scrutinized to make sure another tumor is not being transferred into the wound.

Also, if at the time of the repair, additional tissue is excised so that the repair encompasses an entire cosmetic unit, this additional tissue should be sent for pathological examination, since in patients with severe sun damage, subclinical tumor may be present only a few millimeters away from the tumor free wound. If this is not done, tumor may be incorporated into and buried by the repair, potentially resulting in disastrous consequences.

Although many skin cancers can be managed in a simple manner by a single physician, there are times when a multidisciplinary approach is required to rid the patient of their neoplasm and reconstruct the resulting defect.<sup>66</sup> Such an endeavor may involve a number of specialists including a Mohs surgeon, a head and neck surgeon, a reconstructive surgeon, an oncologic surgeon, a neurosurgeon, or a radiation therapist. Such an approach ensures the patient the greatest chance of cure and at the same time offers the optimal cosmetic and functional outcome. For a number of years now, at the Medical University of South Carolina, we have used such an interdisciplinary approach to manage patients with difficult and aggressive skin neoplasms. Some examples of this interaction between the Mohs surgeon and other specialists will be presented later in this article.

## BIOPSY

Ideally, a biopsy should always be done before definitive therapy is rendered. However this may not always be feasible. If definitive therapy is delivered at the time of initial

contact, an adequate specimen should be sent for pathological examination not only to confirm the clinical diagnosis but also to make sure that the treatment was appropriate and adequate. This is especially important if a blind method of treatment was employed e.g. curettage and electrodesiccation (C&D). A biopsy is mandatory if the diagnosis is in question; a major surgical procedure is planned; or the patient is being referred for radiation therapy. In addition to confirming the diagnosis, multiple biopsies may be beneficial in planning surgery or radiation therapy if the extent of the tumor is in question.

The type of biopsy performed will depend on the lesion, the type of information desired and the anticipated treatment planned. Curettement, as a biopsy technique, is to be discouraged because other than allowing a diagnosis to be made, it does not allow one to determine the overall histology of the lesion; a variable which may be important in determining the most appropriate treatment. Most commonly shave biopsies and punch biopsies are used to evaluate NMSC, however, if one wished to distinguish between a KA and SCC or distinguish pseudoepitheliomatous hyperplasia from a SCC or diagnose perineural involvement, an incisional or excisional biopsy will be required. If one anticipates doing C&D, a deep punch biopsy or incisional biopsy is contraindicated since the resultant wound will interfere with effective curettage.<sup>67</sup> Superficial shave biopsies are adequate for evaluating exophytic lesions; however, flat lesions require a deep shave excision or punch biopsy in order to obtain a correct diagnosis.

## IMPORTANT VARIABLES IN MANAGING NON MELANOMA SKIN CANCER: AGE AND COSMESIS

Although age and cosmesis may be independent factors in deciding on the best management of a NMSC, the two often go hand in hand. As noted above, age alone should not be used as a reason for delivering inappropriate or non-curative treatment. However, there

are older patients who may not want to undergo an extensive surgical procedure and/or reconstruction or who are too fragile to undergo surgery. Such patients may be best managed by radiation therapy.

Aged skin is more forgiving and the elderly often are less concerned about the cosmetic outcome and thus are more accepting of the hypopigmented scar seen with cryosurgery and C&D. On the other hand, younger patients often demand an imperceptible scar that can only be achieved with excision and repair of the defect. All patients should be offered the modality with greatest closure for cure and best cosmetic outcome however.

#### *Number of Lesions*

In some instances, the number of lesions present may dictate the treatment chosen; all other factors being equal. For example, a patient with 20 superficial multicentric BCCs (SMBCC) of the back may best be managed by C&D or cryosurgery rather than excision.

#### *Size of the Lesion*

The size of the lesion may not only be important in terms of cure but also in terms of cosmetic outcome and the complexity of the modality used to treat the patient. A SMBCC of the trunk, if excised, might require grafting; however, it might be successfully treated with topical 5-FU or cryosurgery with as good or better cosmetic outcome.

In terms of cure, 2 cm appears to be the critical size at which the cure rate for invasive BCCs and SCCs<sup>68,69</sup> declines significantly and for SCCs the size at which the incidence of metastases increases significantly.<sup>37,38</sup>

In general, blind techniques such as cryosurgery and C&D should only be used for invasive lesions  $\leq 1$  cm in diameter. Radiation therapy may be used for lesions  $> 1$  cm in size, but a margin of clinically normal skin  $\geq 1$  cm should be included in the field of irradiation because of the unpredictable subclinical spread of these larger tumors.<sup>68</sup> In general, Mohs micrographic surgery (MMS) or excision is the preferred method of treat-

ment for these larger lesions.

#### *Distinctness of Tumor Borders*

Basal cell carcinomas and SCCs with ill-defined borders on histological examination often exhibit tumor growth characterized by small tumor islands, strands of tumor infiltrating the connective tissue, and single cell invasion of the tissue. The stroma may be sclerotic. This growth pattern as regards BCC has been referred to as an "aggressive growth pattern" since it is these tumors which often: (1) recur after routine treatment;<sup>70-76</sup> (2) involve the surgical margins after routine surgical excision;<sup>77</sup> and (3) behave in a biologically aggressive fashion.<sup>72-74</sup> Basal cell carcinomas exhibiting this growth pattern clinically often are flat or plaque-like in contrast to the typical nodular BCC which is exophytic and well-delineated and which on histological examination demonstrates large tumor islands with broad pushing borders. In the past and still today, many pathologists refer to the "aggressive growth pattern" BCC as a morpheiform or sclerosing BCC. Although there is a clinicopathological entity known as the morpheiform BCC, there is no reason to separate it from the other BCCs demonstrating an "aggressive growth pattern."

Because of unpredictable subclinical spread<sup>70-77</sup> and because blind techniques (e.g., C&D) are often inadequate in treating "aggressive growth pattern" BCCs and infiltrating SCCs,<sup>78</sup> such tumors should be managed by surgical excision with histological control, preferably of the Mohs type. Radiation therapy may be used to manage these lesions also but the chance of cure is less and as regards the classical morpheiform BCC, the cosmetic outcome may be poor.<sup>79,80</sup> Pre and post-treatment biopsies may be needed to help plan and assess the effectiveness of the radiation therapy.<sup>80</sup> Including a margin of  $\geq 1$  cm in the treatment field is generally recommended when employing radiation therapy.<sup>79,80</sup> This would also be true if a routine surgical excision was employed.

#### *Histology*

The prognostic implications of histology and



its importance in planning treatment has already been discussed in some of the preceding sections; however, there are still several areas which require discussion.

Knowing the importance of histology in planning treatment, it is no longer acceptable to receive a pathology report which simply says "basal cell carcinoma" or "squamous cell carcinoma." It is mandatory that a full description of the histology of the tumor be given including its growth pattern characteristics, the presence of sclerosis, lymphatic invasion, and follicular involvement, the depth of invasion, and with a SCC, the degree of differentiation. Moreover, when the diagnosis is listed, the subtype of the tumor should be given e.g. aggressive growth pattern BCC.

Superficial multicentric BCCs not uncommonly recur after routine surgical excision either because of marginal involvement, documented by pathological examination, or falsely "clear margins" because of the inadequacy of routine surgical pathology in assessing the adequacy of the excision.<sup>71</sup> Superficial multicentric BCCs often send out microscopic strands of tumor which may not be detected by routine surgical processing. For this reason, MMS, with its ultimate in histological control, is the treatment of choice for recurrent SMBCCs.

Follicular involvement may be seen in association with SMBCC and Bowen's disease (SCC in situ). When present superficial means of destruction such as topical 5-FU, C&D, and superficial cryosurgery will not completely eradicate the tumor. In such instances, routine surgical excision or MMS should be used to manage the lesion.

Metatypical BCCs, because they often recur, behave aggressively, and exhibit extensive subclinical spread, and because they may metastasize,<sup>81-84</sup> should not be managed by blind techniques but should be treated by MMS or routine surgical excision.

If the stroma of the tumor is sclerotic, curettage will be ineffective in eradicating tumor nests embedded within it.<sup>69,85</sup> Curettage will

also be ineffective if the tumor has penetrated deeply into the dermis or subcutaneous fat.

BCCs and SCCs showing perineural spread are best managed by MMS because of the superior histological control achievable with this technique.<sup>28,86-88</sup> At times, this may require multidisciplinary cooperation<sup>87</sup> and adjunctive post-operative radiation. Blind techniques such as ED&C and cryosurgery are unacceptable in the management of such tumors and radiation alone is unlikely to cure such patients, and should be used as the primary form of treatment only in patients who are inoperable or poor surgical risks.<sup>89,90</sup>

Poorly differentiated SCCs should not be managed by ED&C and curettage and are best managed by routine surgical excision or MMS.

Although controversial, many radiation therapists think that radiation therapy is contraindicated in verrucous carcinoma. There have been a number of reports in which the verrucous carcinoma initially appeared to respond to treatment but subsequently recurred, grew rapidly and metastasized. Rebiopsy revealed a marked anaplastic transformation.<sup>91,92</sup>

#### *Anatomical Location*

As noted under "Modes of Spread," certain anatomical locations are associated with a significant risk of tumor recurrence.<sup>28,83,93-101</sup> Such tumors should not be managed by blind techniques but are best treated by routine surgical excision or MMS, preferably the latter. In some of these high risk areas where cosmesis is a concern (e.g., nasal ala, eyelid), there have been those who have proposed that in older patients radiation therapy be used in order to spare healthy tissue and avoid the need for reconstructive surgery. In some instances, this may be acceptable if the chances of cure approach those of surgery.

On the distal nose and scalp, BCC may hide between follicles or bud off them thereby escaping the curette. Also, on the distal nose the dermal collagen is very compact and tumor may be so embedded in it that the

curette cannot extirpate the tumor nest.<sup>102</sup>

In order to be effective, one must have a relatively firm, immobile surface on which to perform curettage. For this reason, effective C&D may be difficult to perform in areas such as the eyelid, lip, and penis.<sup>102</sup>

Cryosurgery is usually not used to treat scalp lesions. Because the scalp is so vascular it is difficult to obtain low enough temperatures to destroy tumor.<sup>103</sup> Aggressive cryosurgery on the legs is associated with prolonged healing, a significant risk of infection, and poor cosmesis.<sup>103</sup>

In general, the trunk and extremities tolerate radiation therapy poorly and the cosmetic outcome is usually inferior to that achievable with cutaneous malignancies of the head and neck.<sup>80</sup>

The cosmetic concerns of the patient as it relates to the location of the tumor may also dictate the modality of treatment selected.

In certain anatomical areas (e.g. nail bed, eyelid) it is mandatory to minimize the loss of healthy tissue if function is to be preserved. In such circumstances, MMS is the treatment of choice.

#### *Pre-existing Conditions*

If the tumor arises in a pre-existing lesion e.g., a burn scar, it should be managed either by routine surgical excision or MMS.

#### *Primary Versus Recurrent Tumor*

As noted under "Modes of Spread," a SCC which is recurrent is much more likely to metastasize.<sup>37,38</sup> Thus it is mandatory to rid the patient of their tumor. MMS is the treatment of choice in such circumstances. The only alternative which is likely to be curative is routine surgical excision. Numerous studies have demonstrated the difficulty in managing recurrent BCCs with routine treatment modalities and the superiority of MMS.<sup>104-107</sup> These tumors are often ill-defined, exhibit an aggressive growth pattern; are embedded in a sclerotic matrix which defies the curette, demonstrate extensive subclinical spread; and send out microscopic strands which may

escape routine pathological examination.

"Field-fire" BCC, in which multiple tumors arise in a circumscribed area, over a period of time, may in some instances represent recurrent BCC whereas in other cases it may be an example of a carcinogenic field effect. Whichever the case, such tumors are best managed by MMS. The resultant wound should be grafted or allowed to heal by second intention.

#### *The Incompletely Excised Basal Cell Carcinoma*

There have been several studies which showed that only one-third of incompletely excised BCCs recur.<sup>108,109</sup> and that when incompletely excised BCCs are re-excised only 50 percent of the specimens contain tumor.<sup>109,110</sup> Based on this information, some clinicians in the past have elected not to re-excise incompletely removed BCCs but to simply follow the patient. Fortunately this is a less common practice today. There are several problems with not reexcising an incompletely removed BCC and a logical explanation for some of the observations of these early investigators.

First of all, it is likely that in many instances, despite marginal involvement, the tumor had been narrowly but completely removed.<sup>108,111</sup> In those cases where reexcision did not reveal any residual tumor, it is likely that only a small amount of tumor was present in the specimen and this was missed by routine pathological examination.<sup>110</sup>

Secondly, in contrast to the above studies, other studies have demonstrated a recurrence rate as high as 86 percent for incompletely excised BCCs.<sup>112,113</sup>

Thirdly, one cannot rely on a patient to detect a recurrence nor can one assume the patient will be steadfast in returning for follow-up evaluation. Thus, by the time the recurrence is discovered, the tumor might be quite advanced. Moreover, if the surgical defect was repaired with a flap, it may be years before the tumor surfaces, and by then the spread beneath the surface may be quite extensive and the consequences disastrous.

For all of the above reasons, we agree with those authors who suggest that all incompletely excised BCCs be reexcised.<sup>113-115</sup>

## TREATMENT MODALITIES

### *Curettage and Electrodesiccation (C&D)*

Although simple in concept and execution, to be effective C&D must be properly applied and performed.<sup>116,117</sup> In performing this procedure, there is no set number of times which the curettage and electrodesiccation must be repeated. Once a healthy base is achieved, the procedure may be terminated. Unnecessary repetition will result in a poorer cosmetic outcome.<sup>118</sup> A 2 to 5 mm margin of clinically normal skin should be included in the area treated to destroy subclinical tumor extensions.<sup>67,69,119</sup> When completing the procedure, a small curette should be employed to remove any remaining small nests of tumor cells.<sup>116,119</sup> Curettage and electrodesiccation offers the advantages of being easy to learn and perform; it is quick and suitable for the patient with multiple lesions. However, it has the disadvantages of leaving a wound which requires four to six weeks to heal, often leaves a hypopigmented scar, and, especially in young adults may leave a hypertrophic scar. Fortunately, however, hypertrophic scars will soften and flatten with time and are responsive to intralesional steroids. Thus, the cosmetic result improves with time.<sup>117</sup> On the nasal tip, a depressed scar may result from C&D. Curettage and electrodesiccation performed near mucocutaneous junctures (e.g., eyelid, lip, nasal alar rim) may result in a cosmetic deformity (e.g., notching of the nasal ala, ectropion). When applied inappropriately, C&D may result in buried tumor which at the time of definitive surgery may exhibit extensive subclinical spread.<sup>120</sup>

Curettage and electrodesiccation is best suited for small ( $\leq 1$  cm), well-delineated, superficially invasive (upper dermis), exophytic lesions, which are not located in areas at high risk for recurrence (see preceding discussion). Histologically, BCCs so treated should show large, well-demarcated tumor

islands with broad pushing borders. Squamous cell carcinomas managed by C&D should be well-differentiated.<sup>69, 121,122,123,124,125</sup> As noted earlier, C&D is ineffective for tumors which (1) involve the follicles, (2) extend deep into the dermis or subcutaneous fat, (3) are enmeshed in a sclerotic stroma, (4) are recurrent, (5) demonstrate perineural or lymphatic involvement, (6) are in an area which cannot be properly immobilized, (7) exhibit infiltration of the dermis by single cells and/or tumor strands, and (8) are poorly differentiated (SCC).

Superficial multicentric BCCs and in situ SCCs, without follicular involvement, which are  $> 1$  cm in diameter are amenable to C&D.

In properly selected patients one can anticipate five-year cure rates in excess of 90 percent. The recurrence rate for head and neck lesions has been reported to be higher than that for tumors of the trunk and extremities.<sup>79,117</sup> This is most likely due to two factors: (1) the high risk areas of the head and neck (e.g., embryonic fusion plates,<sup>102</sup> and (2) a greater preponderance of superficially invasive lesions on the trunk and extremities.<sup>126</sup>

Curettage, used alone, decreases the risk of hypertrophic scarring but not hypopigmentation, and yields a lower cure rate.<sup>127,128</sup>

### *Cryosurgery*

Although at first glance cryosurgery would appear to be simple and easy, it too requires the proper skills and equipment to be effectively executed. To manage BCCs and SCCs, one needs either a cryoprobe or liquid nitrogen spray unit and must have equipment to monitor tissue temperature.<sup>103</sup> Discs and cotton tipped swabs cannot be used to treat even the most superficial of cancers. In general, a double freeze-thaw cycle with a tissue temperature of  $-50^{\circ}\text{C}$  is required to ensure the destruction of most tumors.<sup>29</sup> However, for tumors at high risk for recurrence, a triple freeze-thaw cycle may be necessary. For SMBCCs and SCC in situ (without follicular involvement), a single freeze-thaw cycle is usually curative.<sup>103, 130, 131</sup> For treating most



tumors, it is important to monitor tissue temperature; however, for SMBCCs and SCC in situ (without follicular involvement) and for tumors overlying cartilage and bone, this is usually not necessary.<sup>103,130,131</sup> In the latter instance, one can freeze until the tumor becomes fixed to the bone or cartilage is frozen through to the opposite side.<sup>103</sup> As with all modalities, a margin of clinically normal skin (usually 5 mm) is included in the treated area to destroy subclinical extensions of the tumor.<sup>129</sup> To manage larger or more difficult lesions, cryosurgery may be combined with debulking<sup>103,132</sup> or curettage<sup>132,133</sup> (which also helps delineate the extent of the tumor). Large lesions sometimes require treatment in segments, with treatment extending over several weeks.<sup>103</sup> Lesions at high risk for recurrence may require pre-treatment and post-treatment biopsies to properly treat the patient and ensure the adequacy of treatment.<sup>132</sup>

In recent years, cryosurgery has been advocated in the management of small ( $\leq 1$  cm.) nodular BCCs of the eyelids, since it spares normal tissue (especially the tarsal plate and lacrimal ducts) and obviates the need for reconstruction.<sup>103,134,135</sup> Cure rates as high as 97 percent have been reported; however, for tumors  $> 1$  cm in size or which are recurrent, the cure rate drops precipitously, and reconstruction may be required.<sup>134,135</sup> Cryosurgery is not suitable for deeply invasive lesions or lesions of the fornix.<sup>134</sup>

Cure rates for primary BCCs and SCCs  $\leq 2$  cm in size have been reported to be as high as 98 percent, but in some of these studies, follow-up was of short duration and few details were given.<sup>103,130,131,133,136,137</sup> It is obvious, however, that in order to achieve these high cure rates, careful patient selection and proper execution of the procedure are mandatory. Superficial multicentric BCCs and in situ SCCs (without follicular involvement)  $> 2$  cm. in size have been effectively managed by cryosurgery.

Cryosurgery may also be used for palliation.<sup>103</sup>

The cosmetic outcome of cryosurgery in

general is similar to that of C&D, i.e., a hypopigmented scar.<sup>138</sup> However, as with C&D, hypertrophic scars may occur on the chest and back and depressed scars may occur on the nose and cheek.<sup>103,138</sup> The major disadvantage of cryosurgery compared to C&D is that effective cryosurgery requires more skill and experience and cryosurgery is associated with more morbidity (swelling, blistering, weeping and oozing).<sup>138</sup>

Other complications include: (1) neuralgia and neuropathy (usually reversible) when lesions overlying superficial nerves are treated (e.g., fingers),<sup>138</sup> (2) notching of the nasal alar rim, eyelid, vermilion of the lip, or ear,<sup>103,134,135,138</sup> (3) pyogenic granulomas,<sup>138</sup> (4) infection,<sup>138</sup> (5) transient hyperpigmentation,<sup>138</sup> (6) migraine-like headaches when the scalp,<sup>138</sup> forehead or temples are treated, (7) fever,<sup>138</sup> (8) permanent loss of the eyelashes,<sup>134,135</sup> (9) ectropion,<sup>134,135</sup> (10) damage to the lacrimal ducts<sup>134,135</sup> and (11) buried tumor. The problems associated with treating lesions of the lower legs have already been discussed previously.

The indications and contraindications for cryosurgery are similar to those for C&D, i.e., it is best suited for small primary nodular BCCs and well-differentiated SCCs which are superficially invasive and not located in areas at high risk for recurrence. However, there are some cryosurgeons, with skill and experience, who feel some high risk lesions unsuitable for C&D can be effectively managed by this modality.<sup>103,132,133,134,135,138</sup> Cryosurgery is contraindicated in patients with cold intolerance, blood dyscrasias, dysglobulinemias, autoimmune disease, pyoderma gangrenosum, or who are on immunosuppressives or renal dialysis, or if the tumor is fixed to cartilage or bone.<sup>138</sup>

### *Radiation Therapy*

Radiation therapy is used much less today than in years gone by for the management of NMSC. There are a number of reasons for this. One reason is that dermatologists are no longer trained in administering radiation ther-

apy. Another reason is the overall better health of older individuals which allows them to undergo major surgery. Moreover, there have been great advancements in surgical techniques which allow the removal of skin tumors and reconstruction of the defect. Finally, advancements in anesthesia and supportive care have allowed older and more frail patients to undergo surgery successfully.

To be effective, the radiation therapist must be knowledgeable in the principles of radiation therapy for NMSC and must be thoroughly acquainted with the clinical and pathological aspects of the tumor being treated. In some lesions, special machines, techniques and molds may be required. All lesions to be treated should be biopsied. Not only will this confirm the clinical impression but also tells the therapist about the histology of the lesion and its depth of penetration. A biopsy may also permit the detection of other important features such as perineural involvement and multiple biopsies can be used to map out the lesion.<sup>80</sup> The treatment implications of some of these findings has been discussed in preceding sections of this paper.

The major advantage of radiation therapy is that it spares normal tissue. Thus radiation therapy is often advocated for managing tumors of the nose, ear, and periocular area because it obviates the need for reconstructive surgery and spares the lacrimal apparatus.<sup>80,139,140,141</sup> Radiation therapy also can be used in elderly patients or debilitated patients too frail to undergo surgery or in patients who refuse surgery.<sup>80</sup> It can also be used to palliate inoperative tumors.<sup>142</sup> Radiation therapy also may be used post-operatively as an adjunct if the patient is considered at high risk for recurrent disease or occult nodal metastases are suspected.

In general, radiation therapy should only be used in older individuals because of the slight risk of inducing another cancer and because with the passage of time, the risk of radiation necrosis increases and the cosmetic result deteriorates.<sup>79,80,139,140,141,142,143</sup> After nine to 12 years, only 50 percent of patients treated with

orthovoltage radiation have a good cosmetic outcome. The treatment site often is atrophic, hyperpigmented and hypopigmented, and covered with telangiectasias.

Fractionation provides for a better cosmetic outcome and also decreases the risk of radiation necrosis, especially when treating over bone or cartilage.<sup>80,139,140,141,143</sup>

Currently, radiation therapists use photons, electrons and orthovoltage x-rays to manage most NMSCs. High energy electron beam is especially useful for lesions overlying bone or cartilage because the radiation can be delivered in a more homogenous manner and the dose falls off rapidly so there is less risk of damaging underlying structures. Electron beam may also yield better cosmesis, although mild scarring and patchy pigmentary alteration may occur.<sup>144,145</sup>

Tumors > 5 cm in size,<sup>80,144</sup> those involving bone,<sup>48,80</sup> and those arising in scars or areas of prior irradiation are best managed surgically,<sup>80</sup> because the risk of recurrence is high as is the risk of complications.

If the margins of excision are involved with tumor, it is preferable to re-excise the lesion, if possible, rather than referring the patient for radiation therapy. In a situation such as this, one may be dealing with a tumor with significant subclinical spread and it may be difficult for the radiation therapist to effectively treat the lesion.

The philosophy that a tumor can be irradiated and if this fails it can always be cured surgically is to be discouraged.<sup>141</sup> Following radiation therapy, tumor can be buried and spread extensively subclinically before being detected.<sup>146</sup> Moreover, a previously irradiated area is predisposed to problems with wound healing.

As might be anticipated, cure rates for BCCs and SCCs will vary according to the size, location, and histology of the lesion and whether or not it is recurrent. Cure rates for primary BCCs and SCCs which are < 2 cm. in size, don't exhibit an aggressive growth pattern, and are not in high risk locations, have been reported to be in excess of 90 percent.<sup>79,80,123,139</sup> Tumors which are at significant

risk for recurrence are best managed by surgery.

As noted previously, the cosmetic outcome of radiation therapy on the trunk and extremities is often poor and care needs to be taken when treating lesions of the dorsa of the hands and feet to avoid damage to deeper structures.<sup>139,143</sup> Scalp lesions are generally not treated by irradiation because of the permanent alopecia which occurs.<sup>143</sup>

Following irradiation, erythema, oozing, crusting, itching, burning and paresthesias are not uncommon. The area may ulcerate. Healing may require weeks to months.<sup>80</sup> Although some tumors melt away rapidly, others may require six to 12 months to disappear.<sup>80,139,142</sup> A tumor persisting beyond this time should be rebiopsied.<sup>80</sup> If massive and persistent necrosis occurs, one should suspect that more tumor was present than was clinically evident and that tumor might still be present. Under these circumstances, the patient should be rebiopsied.

Complications of radiation therapy, other than those already mentioned, include: (1) scarring; (2) notching of the eyelids, lips, and alar rims; (3) scarring of the lacrimal duct with resultant epiphora; (4) radiation necrosis (one to five percent incidence); (5) keratitis; (6) cataracts; (7) alopecia; (8) perforation of the eye; (9) mucositis; (10) comedones; and (11) chronic radiation dermatitis.

Although offering a number of advantages, one of the greatest disadvantages of radiation therapy, when treating a cancer that could have been handled equally as well in the office by another method, is the cost and necessity for multiple visits. Thus, patient selection is extremely important when employing this modality.

### *Excision*

Surgical excision may be used to manage the smallest and simplest of tumors or the largest and most difficult ones. When MMS is not readily available, it is the treatment of choice for lesions at high risk for recurrence. Excision offers the advantages of histologic con-

trol, rapid healing, and optimal cosmetic and functional results. However, compared to procedures such as C&D, it is more time consuming, less suitable for multiple lesions, sacrifices normal tissue, requires more training and skill, and may necessitate reconstructive surgery.

Because the surgical specimen removed by simple excision is not sectioned in its entirety, excisional surgery offers inferior histologic control when compared to MMS which includes complete pathological examination of the entire peripheral and deep margins of the specimen(s). Mohs micrographic surgery is also more tissue sparing because rather than taking arbitrary margins of normal adjacent skin, the tumor is systematically mapped and traced out.<sup>149</sup> Because the histologic control with surgical excision is inferior to that of MMS, one observes a cure rate which is inferior when dealing with high risk lesions (recurrent lesions, BCCs with an aggressive histology, SMBCCs, in situ SCCs, and lesions in high risk areas).<sup>68,71,74,76,79,104,148</sup>

Excisional surgery often can be carried out in the office or in an outpatient setting, however, the removal of larger and more difficult tumors may require general anesthesia.

The recommended margins for excision of a BCC or SCC have varied from author to author and sometimes have been based more on subjective impressions rather than objective findings. Perhaps the most systematic and objective investigation of this question was carried out by Zitelli and co-workers.<sup>151,152</sup> For sharply demarcated non-morpheaform BCCs  $\leq 2$  cm in diameter, 4 mm margins of clinically normal skin were adequate to achieve clear margins in 98 percent of lesions. For lesions  $> 2$  cm in size, the sub-clinical spread was so variable that rigid recommendations could not be made.<sup>151</sup> For well-differentiated SCCs  $< 2$  cm in size and not located on the scalp, ears, lips, eyelids or nose and not invading the subcutaneous fat, 4 mm margins were adequate.<sup>152</sup> However, tumors  $\geq 2$  cm in size, which were in one of the high risk areas mentioned above, or which



invaded fat or which were not well-differentiated, required 6 mm margins.<sup>152</sup> For most small primary tumors (BCC and SCC) of the skin, invasion of the fat is uncommon. Therefore, if the excision is carried well into the subcutaneous fat, a clear deep margin should be assured. This will also facilitate closure of the defect. Following the above guidelines set forth above, authors have reported cure rates as high as 98 percent with excisional surgery.<sup>123,125,148</sup>

As mentioned in previous discussions, it is mandatory that a tumor-free wound be achieved before repairing the defect, especially if a flap is used. The consequences of opening up tissue planes and burying tumor can be disastrous. For simple excisions and repairs, a specimen sent for routine pathological examination will suffice, but for difficult excisions and complex repairs, intraoperative frozen sections should be utilized. If there is any question regarding the completeness of tumor removal, the defect should be repaired with a thin graft (if feasible). Also in patients with a history of numerous skin cancers, the donor site for a graft or flap should be scrutinized carefully lest tumor be transferred into the wound.

Complications of excisional surgery include hematoma formation, swelling, pain, hyperpigmentation, hypopigmentation, failure of the flap or graft to survive, ectropion, and less than ideal scarring. In patients with a less than satisfactory outcome, it should be remembered that cosmesis improves with time and scar revision is possible.<sup>148</sup>

#### *Mohs Micrographic Surgery*

Over 50 years ago, Frederic E. Mohs, at the University of Wisconsin, developed a technique to manage difficult skin neoplasms.<sup>154</sup> That technique is now known as Mohs micrographic surgery (MMS). Since its inception, the technique has undergone a number of modifications. In the early years of the procedure, a zinc chloride fixative was always applied before the tumor was excised. However, this so-called "fixed tissue technique"

had a number of drawbacks, including severe pain, poor quality histologic sections, an inability to do immediate repairs, and the limited ability to remove more than one layer of tissue per day. However, in the 1970's, Drs. Mohs, Tromovitch and Stegman<sup>107</sup> began to experiment with the "fresh-tissue technique." With this modification, the zinc chloride fixative was omitted and local anesthesia was used. This allowed the procedure to be less painful, to often be completed in a single day, and also yielded better histological sections and allowed for immediate repairs. It was also found that the cure rate was just as high as with the "fixed-tissue technique." Today, the fresh-tissue technique is almost exclusively employed by Mohs surgeons.

In the early years, Dr. Mohs advocated letting most wounds heal by second intention. Although the results were often impressive, this approach sometimes necessitated prolonged wound care and sometimes did not yield optimal cosmetic and functional results. Once the "fresh-tissue technique" was introduced, immediate repairs became possible. Consequently, Mohs surgeons began to advocate repairing Mohs surgical defects. This change in philosophy resulted in a number of benefits: (1) faster wound healing; (2) better cosmetic and functional results; (3) wider acceptance of the procedure; and (4) an interdisciplinary approach to the management of difficult skin neoplasms in which the Mohs surgeon teams up with a reconstructive surgeon and/or oncologic surgeon to rid patients of their cancer and provide the best cosmetic and functional outcome.

Many of the indications for and advantages of MMS have been reviewed in preceding sections as well as elsewhere and will not be repeated in detail here, other than to say that when dealing with high risk lesions or lesions in locations where the maximum preservation of tissue is essential, MMS offers the highest chance of cure while maximally preserving healthy tissue.<sup>66,149</sup>

The procedure is generally performed as an outpatient using local anesthesia. However,

extensive, deeply invasive tumors may require the help of other surgeons and the use of general anesthesia. Because it is performed under local anesthesia as an outpatient, it is cost-effective and extends operability to patients who are poor surgical risks. In many cases, the tumor can be removed and the defect repaired in the same day. Extensive defects may require the help of a reconstructive surgeon. When this can be anticipated, a coordinated effort is planned ahead of time. However, even when the surgical defect ends up much larger than anticipated, the delay between the completion of Mohs surgery and reconstruction is usually not prolonged.

In performing MMS, the Mohs surgeon serves both as the surgeon and the pathologist. The laboratory where the tissue is processed and microscopically examined is located in the surgical suite.

With MMS, once the obvious tumor has been removed, a thin (1-2 mm thick) layer of tissue is removed. This layer of tissue is subdivided, color-coded, and mapped and submitted for microscopic examination. Any areas showing residual tumor are marked and additional tissue is taken from this area(s), color-coded, mapped and examined microscopically. The process is repeated until a tumor-free wound is achieved. For BCCs and SCCs, frozen sections can be used, but for some tumors (e.g. melanoma), the tissue must be processed overnight to yield permanent sections. Regardless of whether frozen sections or permanent sections are rendered, unlike routine surgical specimens, the entirety of the peripheral and deep margins of the submitted tissue are examined, thus explaining the high cure rate of MMS. Once a tumor-free wound is achieved, the repair of the surgical defect can be addressed. The same conservative approach used in managing routine surgical defects also applies to Mohs surgical defects.

Complications associated with MMS are similar to those associated with routine surgical excision. As with all procedures, MMS has several disadvantages. When dealing with

tumors with extensive subclinical spread, the procedure may become prolonged and tedious for both patient and surgeon. The other major disadvantage is that it is not always readily accessible and requires special equipment (cryostats) and highly trained personnel (histotechnologists).

The cure rates for MMS for primary and recurrent BCC is 99.8 percent and for SCC is 98.8 percent.<sup>105</sup>

### *5-Fluorouracil*

Topical 5-fluorouracil (5-FU) is indicated only for the treatment of actinic keratoses, and SCC in situ and SMBCC without follicular involvement.<sup>155,156,157,158</sup> It is contraindicated in invasive SCCs and BCCs since it is ineffective and may bury tumor, allowing extensive subclinical spread of the carcinoma before it is detected.<sup>159</sup> When treating large lesions, where only a small biopsy has been obtained, it should be remembered that limited sampling is subject to sampling error and that subclinical invasive disease may be present and consequently buried. Thus, lesion selection is extremely important. Only the five percent concentration of 5-FU is suitable for treating SCCs in situ and SMBCCs. To achieve acceptable cure rates, treatment must be continued for six to 16 weeks,<sup>155,156,157,158,160</sup> and, at times, occlusion may be required to achieve the desired therapeutic effect.<sup>155,156,157,158</sup> During treatment, the patient may suffer significant discomfort, and the treated area will become red and eroded and will weep and ooze. Sequelae from the use of topical 5-FU include hyperpigmentation, hypopigmentation, scarring, prolonged erythema, onychodystrophy, onycholysis, and telangiectasias.<sup>155,157,158</sup> Cure rates of 92 percent have been reported for SCC in situ<sup>160</sup> and 95 percent for SMBCC.<sup>156</sup> Although potentially affording a better cosmetic outcome than a modality such as C&D, the need for prolonged treatment and the discomfort associated with treatment make topical 5-FU of limited usefulness in the management of NMSC.

Currently, a sustained release formulation

of 5-FU for intralesional use is being investigated. Injections are given weekly for up to six weeks. In a group of patients with BCCs, the cure rate was 80 percent.<sup>161</sup>

### *Lasers*

Lasers play a limited role in the day to day management of BCCs and SCCs. The carbon dioxide (CO<sub>2</sub>) laser can be used in the focused mode to excise a lesion or in the defocused mode to vaporize lesions.<sup>162</sup> Although theoretically offering an advantage for excising a lesion in a patient on anticoagulants or with a pacemaker, the safety hazards and awkwardness of the CO<sub>2</sub> laser system usually outweigh any benefits, real or imagined. In the defocused mode, the CO<sub>2</sub> may be used in conjunction with curettage to treat SMBCCs, SCC in situ and Bowenoid papulosis (BP).<sup>163,164</sup> Although useful for BP, the cosmetic outcome when treating SMBCC and SCC in situ is no better than that seen with C&D.

The neodymium:yttrium-aluminum garnet (Nd:YAG) laser has been reported to be effective in the management of invasive skin tumors; however, experience to date has been limited and significant scarring can be expected.<sup>165</sup>

### *Photodynamic Therapy*

Photodynamic therapy (PDT) is an experimental form of treatment in which a photosensitizer (e.g., hematoporphyrin) is administered intravenously,<sup>166,167</sup> intralesionally,<sup>167</sup> or topically<sup>168</sup> and subsequently activated by light from an argon pumped tunable dye laser or gold vapor laser. This results in tumor necrosis. Considerable discomfort may occur and the area heals leaving a scar. Although encouraging results have been reported in treating NMSC,<sup>169</sup> there are a number of problems with PDT including the cost and the need for special expensive equipment. Also, intravenous administration of hematoporphyrin is associated with a generalized photosensitivity which may last up to six weeks.<sup>166,167</sup> Work is now going on to find

agents which are effective but don't induce a photosensitive state.<sup>170,171</sup> When the photosensitizer is applied topically, there are problems with penetration of the agent<sup>168</sup> and when the photosensitizer is administered intralesionally or intravenously, there are problems with penetration of the laser beam. This latter problem in part can be overcome by debulking the tumor or using interstitial laser fibers.

### *Interferon*

Intralesional interferon has been used to successfully treat BCCs and SCCs with cure rates as high as 96 percent.<sup>172,173,174,175</sup> The tumor is usually injected thrice weekly for three weeks. Side-effects include discomfort at the injection site, leukopenia, and flu-like symptoms. This treatment is still considered experimental. Like all blind techniques, it carries the risk of incompletely destroying the tumor and allowing its subclinical spread before it is detected. Thus, lesion selection is extremely important. It also has the disadvantages of being expensive and requiring multiple visits.

### *Retinoids*

Both isotretinoin and etretinate have been used in the management of NMSC.<sup>176,177,178,179,180</sup> In general, they are more effective as chemopreventive agents than as chemotherapeutic agents. However, once they are stopped, their protective effect is lost.<sup>177</sup> They have been used to prevent tumor development in xeroderma pigmentosum patients<sup>176</sup> and transplant patients.<sup>177,178</sup> SCCs are more responsive to these agents than are BCCs. Response rates as high as 71 percent have been reported in patients with advanced, recurrent, or metastatic SCCs of the skin.<sup>178</sup> Recently, investigators have used the retinoids in combination with interferon and achieved even higher response rates (93 percent) in patients with advanced cutaneous SCCs.<sup>180</sup> □

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# COMPREHENSIVE CARE OF THE CLEFT LIP AND PALATE PATIENT

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Children born with cleft lip or palate often suffer with a multitude of problems associated with these anomalies. There is a high association of additional congenital anomalies in this population group. In addition to dental, speech, and ear difficulties, there is often abnormal facial growth and occasionally swallowing and breathing difficulties. Because of the multitude of problems a multidisciplinary team usually provides the best care for these patients. The team should consist of a speech pathologist, dental specialists, a cleft surgeon, social worker, psychologist, and geneticist.

Cleft lip and/or cleft palate occurs in one out of 680 births and is the second most common congenital deformity in the human population after club foot.<sup>1</sup> The incidence varies among different population groups as well as the sexes. Native Americans have the highest rate of cleft lip followed by the Asian and Caucasian population with the lowest incidence occurring among African Americans. Males have a higher incidence of cleft lip while females have a higher incidence of cleft palate. Twenty to 30 percent of cleft defects are isolated cleft lips while 35 to 55 percent involve only the palate.

## SURGICAL REPAIR

There is marked variation in the timing of the surgical repair of the cleft lip. However, in the United States, the majority of surgeons favor performing the procedure at eight to 10 weeks of age. Many cleft surgeons repair bilateral cleft lips in two stages with the second side of the defect being repaired six to eight weeks after the first. There are several techniques

used to repair cleft lips. However, all of these include reconstruction of the orbicularis oris muscle, cupid's bow, and a symmetrical vermillion border and a symmetrical nose.

In this country the most widely accepted practice is to surgically close the palate defect by 12 to 18 months of age. This strikes a compromise between maximizing speech development and minimizing facial growth disturbances. Any residual mid-facial bony abnormality is dealt with by maxillofacial surgical techniques later in life. This is the standard promoted by the American Cleft Palate Craniofacial Association.

## AIRWAY OBSTRUCTION

The most urgent of cleft-associated symptoms is fortunately uncommon. Most patients with cleft palate and airway difficulty have the Pierre Robin sequence. This consists of mandibular hypoplasia which leads to a posterior positioning of the tongue (glossoptosis) and quite often a midline posterior palate cleft. Even severe airway obstruction in Pierre Robin often improves markedly by four to six months of age.<sup>2</sup> Usual treatment consists of maintaining a prone position which exploits the influence of gravity on the tongue thus causing it to fall out of the airway and alleviate the symptoms. If the patient has an unacceptable amount of respiratory distress while maintaining these measures, a tracheostomy usually must be performed. Other measures such as nasopharyngeal intubation or glossopexy (lip-tongue adhesion) have questionable efficacy or practicality.

## FEEDING AND NUTRITION

Feeding difficulties are what usually bring the otolaryngologist or speech pathologist to eval-

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Figure 1. Newborn with unilateral cleft lip.



Figure 3. One-year-old child with repaired cleft lip prior to cleft palate repair.



Figure 2. Same child in Figure 1 at age 18 months, 15 months after the lip repair.



Figure 4. Same child in Figure 3 after cleft palate repair.

uate the newborn with a cleft deformity. Because of the inability of the infant to generate effective oral suction, methods to compen-

sate for the oral nasal communication are needed. Most feeding techniques consist of using various devices that permit direct expression of milk into the infant's oropharynx so the child can then initiate a swallow. The nipples are soft and large with a generous opening. The bottles are also soft and consist of disposable bags thus allowing milk to be squeezed from the bottle into the mouth without the child having to initiate sucking. The child is often held upright to allow him to have better control of the flow of fluid before it hits the posterior pharynx and to lessen the chance of nasal regurgitation. Due to the fact that the cleft child swallows a large amount of air when feeding, frequent burping is needed. Feeding is initially laborious and time consuming but it is usually the child's mother

who determines (after much anxiety and consultations by health professionals) exactly what position, bottle type, nipple size, and nipple opening is best for the child. The goal is for the child to maintain proper weight and growth as one would expect of a child without a cleft. Rarely do measures such as gavage feedings and nasogastric tubes need to be implemented on a cleft lip or palate child who has no other deformities.

### **SPEECH**

Cleft of the lip and/or palate has an impact on the patient's ability to correctly enunciate many sounds used in the English language. Positioning of the tongue against the lips, teeth, and palate, and most importantly escape of air from the mouth into the nose are greatly affected by the cleft deformity. Early evaluation, therapy, and careful follow up by speech pathologists experienced in working with cleft palate patients is essential. Fiberoptic evaluation of the nasopharynx and posterior oral cavity appears to be replacing fluoroscopy as the main method of evaluation of velopharyngeal insufficiency (leak of air from the oral to the nasal cavity). Close cooperation and interaction between the surgeon and the speech pathologist is needed to determine when surgical intervention is needed, and what type of procedure would best alleviate the velopharyngeal insufficiency. Post-operative speech therapy will be needed to enable the patient to be successful in the difficult process of reversing old speech habits.

### **DENTAL**

Dental development and hygiene particularly in the very early stages of tooth eruption and deciduous dentition need qualified care and future planning. Occasionally orthopedic devices are needed to assist in the placement of dentition. The orthodontist and cleft surgeon must work closely together to determine proper timing of the alveolar bone grafting as well as to determine the need for surgical advancement of the mid-face to bring about normal occlusion and bony profile.

### **EAR DISEASE**

There is a greater incidence of hearing loss in the cleft palate population than in the normal population. Patients with a cleft palate have an abnormal insertion of the levator palatini and tensor veli palatini muscles to the posterior end of the remaining hard palate instead of across the palate to the opposite side. This is probably the main contributor to impaired eustachian tube dysfunction which is the primary cause of hearing loss in this population. With increasing age the incidence of eustachian tube dysfunctions decreases. However, when compared to the non-cleft population the cleft palate eustachian tube dysfunction runs a protracted course.<sup>3</sup> Clinical examination of the tympanic membrane and insertion of ventilation tubes as needed appear to greatly preclude the need of more comprehensive ear surgery later as the child develops.

### **REVISION SURGERY**

Even the most acceptable scars and soft tissue proportions following the initial lip and nose surgery can change as the child grows and develops. Thus a large number of cleft children undergo secondary surgery when they reach their teenage years. Patients with a cleft lip often have an associated nasal deformity. Though often this deformity is addressed during the initial repair of the cleft lip one does not do a definitive procedure on the nose until most of the facial growth is complete (14 to 18 years of age). The cartilaginous and bony skeleton, alar size, and nasal projection all are addressed in the definitive procedure to correct the cleft nose.

Due to the orolaryngologist's background in soft tissue and bony surgery of the head and neck, airway difficulties, swallowing difficulties, speech difficulties, and otology these specialists are finding a more prominent place on cleft teams. The eventual outcome, however, is dependent on the involvement of specialists versed in the myriad of problems the cleft patients experience. No two teams will be identical, but it is the multidisciplinary concept that ensures the highest quality care for



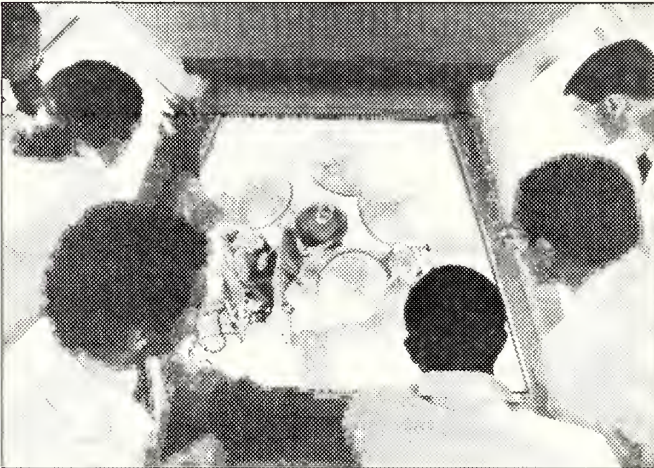
these special patients.



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# OTITIS MEDIA: WHEN ARE TUBES AND/OR ADENOIDECTOMY NEEDED?

JACQUELYN A. GOING, M. D.\*

DANIEL M. ERVIN, M. D.\*\*

Otitis media is not a simple disease, and there is no definitive answer to the title question. Controversy exists as to the timing and methods of treatment of recurrent acute otitis media and chronic otitis media with effusion.

Myringotomy with ventilation tube insertion, which has gained popularity since the 1950s, is now performed on people of all ages but most often in children because of their proclivity to middle ear disease from impaired eustachian tube function, immature immune system, and adenoid bulk. This procedure is probably the most common surgical procedure in children.<sup>1</sup>

Medical treatment prior to consideration for surgical intervention has been the subject of many studies. Various antibiotics have been recommended and evaluated. First-Line antimicrobials for recurrent acute otitis media and chronic otitis media with effusion probably remain amoxicillin or trimethoprim-sulfamethoxazole<sup>2</sup> with amoxicillin clavulanate or second generation cephalosporins commonly applied to treatment failures or refractory cases. However, a recent review article on the use of antibiotics for prevention of recurrent acute otitis media and the treatment of chronic otitis media with effusion concludes that long term benefit has yet to be proven in the latter.<sup>3</sup>

Children with recurrent acute otitis media and chronic otitis media with effusion fall into three general patterns.<sup>4</sup> Some children will have recurrent acute otitis media which responds to antimicrobials and clears with normal hearing and ear examination between episodes. These children may do well with

antimicrobial prophylaxis such as single evening) doses of amoxicillin (20 mg/kg) or of sulfisoxazole (75 mg/kg). Another group may have persistent middle ear effusion which is virtually asymptomatic except for a hearing loss which is often discovered on a school screening audiological examination. A third group of children will have the pattern of recurrent acute otitis media with persistent middle ear effusion and hearing deficit between the acute episodes. It is important to establish the pattern with a careful history and follow-up examination at the end of each course of antibiotics. Recurrent acute otitis media and/or chronic otitis media with effusion do have sequelae such as progression to tympanic membrane retraction pockets, cholesteatoma, or ossicle erosion, and may necessitate extensive ear surgery. Studies have also found an association between the time spent with middle ear effusion during the first three years of life and lower scores on tests of cognitive ability, speech and language.<sup>4</sup>

Surgical intervention, i.e., myringotomy and insertion of ventilation tubes, is designed to evacuate middle ear effusion and facilitate middle ear ventilation while affording time for reversal of middle ear changes and improvement of eustachian tube function. This improves hearing and minimizes progression to chronic otitis media sequelae.

In general, indications for myringotomy and insertion of ventilation tubes are recurrent acute otitis media with persistent middle ear effusion, or chronic otitis media with effusion unresponsive to antibiotics for three months, or frequent recurrent acute otitis media with apparent clearing between episodes with break through of the antimicrobial prophylaxis. The three-month rule is not absolute. Con-

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sideration for earlier placement of tubes is indicated in patients with craniofacial anomalies associated with middle ear aeration impairment such as cleft palate or Down's syndrome. Earlier placement should also be considered in a child with multiple antibiotic allergies and in a child with a sensorineural hearing loss where the addition of a conductive component further impairs functional hearing even with a hearing aid. The presence of an atelectatic tympanic membrane or retraction pocket might also call for more flexibility in timing of ventilation tube insertion.

A national survey of board certified otolaryngologists to ascertain the criteria they considered important indications for myringotomy and insertion of ventilation tube found that the first four indicators were in order of ranking: (1) persistence of fluid for three or more months, (2) speech language delay, (3) presence of bilateral conductive hearing loss of 20 db or more, (4) total number of episodes of otitis media.<sup>1</sup>

There have been a number of studies of the efficacy of adenoidectomy on recurrent acute otitis media and chronic otitis media with effusion. Some studies have shown beneficial effects on the reduction of illness and others have not. Current recommendations are that adenoidectomy be considered if the child needs reinsertion of ventilation tubes, or if a child has symptoms of substantial adenoid hypertrophy with airway obstruction such as snoring or mouth breathing.

As noted, otitis media with effusion remains a complex and controversial topic with differences of opinion concerning therapy not only between different specialties but within otolaryngology itself. Optimal medical approaches and duration of therapy or observation are as yet ill defined. The best treatment plan takes into consideration the well-being of the child

and is cognizant of parental desires and concerns. A child who is chronically ill with recurring infections puts various strains on the overall family situation, aside from time lost from work or sleep and the expense of repeated visits to the family physician or Emergency Department. Such a child with documented poor response to aggressive antibiotic therapy would certainly be a candidate for early ventilation tube placement and usually will do very well. Even when the odd infection develops in spite of having tubes in place, they are generally less symptomatic and more easily controlled.

On the other hand, the child having an asymptomatic effusion with no substantial loss of hearing or speech development problem can safely be observed for some time. In such cases consideration of other factors, such as summertime aquatic activity, can be entertained and the tube placement delayed until a time more convenient for patient and family. As one can readily see, many factors come into play, some outside the realm of medicine or surgery, but the physician must keep in mind the ultimate goal of the healthy child with appropriate hearing and speech development, and constantly strive to minimize the serious long term sequelae of inadequately treated otitis media. □

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# HEARING AIDS: SELECTION, PARAMETERS, & LIMITATIONS

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The ability to hear and interpret speech and language is one of our senses that often is taken for granted and when problems occur, often misunderstood. To properly diagnose and treat hearing loss there needs to be first an awareness among physicians as to the types of hearing loss that commonly occur and an index of suspicion to pursue appropriate evaluation.

When a patient presents to an office with hearing loss, often family assistance is significant in describing the problems or degree of hearing loss for the patient. For most people hearing loss is painless, slow in onset, and the patient may not be fully aware of the problem, whereas family members and co-workers can give more detailed information as to the magnitude of the loss and its impact on daily life.

There are several types of hearing loss accessible to evaluation. Conductive hearing loss is defined as a blockage of sound transmission to the inner ear. The causes of conductive hearing loss are many (Table 1). The treatments are individualized based upon the patient's age, physical findings, and general medical condition. For many conductive hearing loss patients surgery may give marked or total return of hearing to normal levels, such as with cerumen impactions, drainage of serous otitis and repair of otosclerosis. For patients in whom either medical or surgical intervention is not successful or possible due to underlying medical conditions, amplification is an option for rehabilitation of hearing.

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**TABLE 1**

1. Cerumen Impaction
2. External Otitis
3. Tympanic Membrane Perforation
4. Middle Ear Fluid
5. Ossicle Fixation

Sensori-neural hearing loss is defined as a hearing impairment in the inner ear or central auditory connections (Table 2). As with the conductive hearing loss, sensori-neural loss may have a multitude of etiologies and a full history both of the patient and the family is indicated with radiological and metabolic evaluations, as appropriate. In contrast to amplification use with a conductive hearing loss, amplification in sensori-neural hearing loss is often hampered by reduced speech and language discrimination as a result of cochlear or auditory nerve damage. Patients need to be informed of treatment options and expected results with consideration, in some cases, of assistive listening devices (i.e., telephone amplifiers, infra-red TV systems, etc.) or personal amplification in the form of hearing aids.

**TABLE 2**

1. Presbycusis
2. Noise-Induced Hearing Loss
3. Meniere's Syndrome
4. Acoustic Neuroma
5. Hereditary Hearing Loss

Some patients will present a combination of conductive and sensori-neural hearing loss; this type of hearing loss is labeled a mixed hearing loss. Central Auditory Dysfunction is an entity whereby the neurological centers within the brain are impaired. In these patients sound levels are near normal on audiological testing, but poor integration of speech and language understanding is present. Often children or patients with Alzheimer's Disease are felt to have hearing loss when in reality they have Central Auditory Dysfunction. For some, rehabilitation with audiological and speech-language pathology intervention may help.

Research data have suggested that people usually wait 10 to 15 years from the time they first notice some difficulty with their hearing until they obtain a hearing aid. During this time interval a whole spectrum of auditory, psychological, social, emotional and health changes take place in the patient. People are often dealing with "mid-life" crises, thus carrying extra life change baggage, when they first approach the purchase of a hearing aid. The hearing aid is often expected not only to make them hear better but also make their life in general better. If their life does not improve the hearing aid may be blamed.

To evaluate the patient, audiological testing in conjunction with physical examination helps to determine the type of hearing loss, the degree of hearing loss, and to provide a prognosis regarding successful use of amplification.

While 28 million Americans, or roughly one in nine, present a hearing impairment that could be eased by the use of a hearing aid, fewer than one in four utilize such devices. Random surveys of hearing aid dispensers in 1991 and 1992 revealed that otolaryngological referrals accounted for 19.4 percent of amplification purchases while general medical referrals numbered 7.7 percent. In order of rationale for this lack of amplification use individuals usually list the following reasons: fail to seek professional help, inappropriate advice by primary care providers that a hearing aid is not useful, misinformation that their type of hearing loss is not severe enough or

cannot be helped by amplification, unrealistic expectations of the benefits of the hearing aid, cost of a hearing aid, perceived stigma associated with hearing aid use (age or disability), and the reputation of unethical hearing aid dealings.

A hearing aid is defined as a medical device by the Food and Drug Administration and informed consent usually is obtained prior to dispensing. Aids do not restore normal hearing, but do enhance listening capability. Followup is highly desirable after dispensing an aid, and may encompass a variety of audiological services including a 30-day trial period of amplification, amplification orientation, lip reading, counseling, communication strategies, related technological information and support groups. Hearing aids do not solve every listening problem nor do hearing aids perform well in many situations of competitive loud noise, (e.g. a crowded restaurant). Hearing aids do help to make soft sounds louder, help people hear consonants, reduce strain of listening and reduced repetition of conversation. Presently, in the United States 105 manufacturers of amplification provide devices to the market place. Five manufacturers account for more than 70 percent of today's market place. Presently more binaural than monaural fittings take place. In 1992, 1,766,796 hearing aids were purchased in the United States with 60 percent of the users over age 65. In South Carolina, 1992 sales numbered 13,781 representing a four percent increase from 1991 hearing aid sales.

The basic problem of prosthetic use with hearing loss is that in attempting to mirror the function of the human cochlea one must consider that while 60 percent of sound energy in ordinary conversation is contained in low frequency vowel sounds, those sounds convey only about five percent of speech actual information. High frequency consonant sounds contain roughly five percent of speech power, but convey 60 percent of its intelligibility (Figure 3).

Major advancements in electronics, such as vacuum tubes, transistors, and integrated cir-

cuits have provided the necessary technology for major changes in size and features of traditional hearing aids. Audiological procedures leading to maximum hearing aid potential include the use of insert earphones, probe microphone measurements, and individualized prescriptive fitting methods.

Popular acoustic circuit alternatives to conventional amplification include: Class D receivers, active tone control, and directional microphones. These circuit options provide decreased distortion, improved speech to noise ratios and less battery drain.

*Hearing Aid Styles:* In-the-Ear (56.9 percent of the 1992 market sales) and In-the-Canal (25 percent of hearing aid sales in 1992) incorporate a type of virtual reality in their use of incorporating characteristics of the external ear to enhance sounds inside the ear. The scoup like shape of the external ear collects sounds, similar to cupping your hand behind the ear. The canal presents a natural resonance which is altered by the addition of a hearing aid. By plugging the ear with an In-the-Ear (Figure 5) or In-the-Canal (Figure 2) hearing aid the auditory canal is transformed into a resonating chamber where low frequency sounds, like those made by chewing, are grossly amplified. Electronic venting, which provides the feeling of a normal open ear, is presently available from one manufacturer.

*Deep Canal Fittings (Figure 1) :* Twenty-three manufacturers to date are providing deep canal fittings. A reduced "stopped up" feeling is expected. Early models include:

*Earlens:* Developed in 1990, a tiny magnet is affixed directly to the eardrum and held in place by a drop of oil. The earlens amplifies the movements of the eardrum when activated by signals from a small receiver worn on the upper body.

*XP Peritympanic Device:* Introduced in 1993, this tiny, soft, deep-seated device almost touching the eardrum, may be the appropriate

amplification for 30 percent of the hearing impaired population who have certain anatomic or medical preexisting conditions. The quarter-inch wide and quarter-inch long device will cost between \$1,500 and \$2,000 or approximately 20 percent more than conventional hearing aids.

*Tympanette:* In this micro canal hearing aid, the volume is set by the dispenser and not adjusted by the user. Removal of the hearing aid is accomplished by tugging on a transparent cord.

*Behind-the-Ear:* Behind-the-Ear (BTE Hearing Aids) are air conducting hearing aids employing tubing to an individually molded "ear mold" routing the amplified sound to the ear canal. In general, BTEs are more powerful, based on the battery size and circuitry available. For people with severe to profound hearing loss often this extra power is needed for amplification. BTE hearing aids accounted for 16.2 percent of market sales in 1992 in the United States. Several adaptations of this style of hearing aid are now available.

*Extendear:* The unit combines an FM wireless hearing system for school or work availing the possibility of overcoming competitive noise distance (100 ft.), and reverberation in a personal hearing aid (Figure 4).

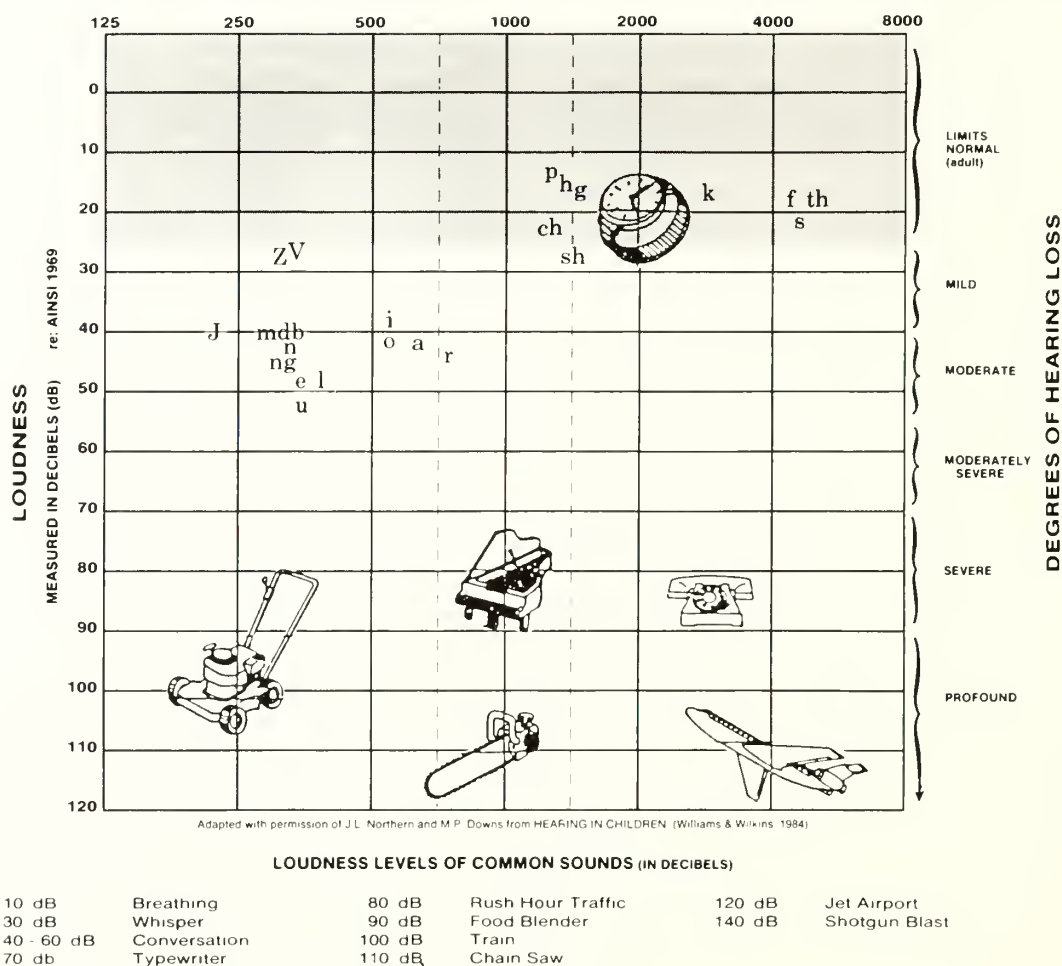
*Programmable Multi-memory Hearing Aid:* Currently marketed by 17 manufacturers in the form of Behind-the-Ear, In-the-Ear, and In-the-Canal styles. The number of memories available range from two to eight with the availability of remote control for memory switching on most. Each Multi-memory Hearing Aid has its own unique feature making comparison among the devices difficult. In fitting this kind of hearing aid, the audiologist must first determine if the candidate is an appropriate user. The need for multi-memories is highly questionable if the user notices no performance difference among memories or uses only one memory exclusively. The fear





### Figure 2. Canal Hearing Aid

With the promise of fully digital (realtime



**Figure 3. Percent Frequency Contribution toward speech understanding**



Figure 4. Extendear transmitter and receiver.

analysis) hearing aids already marketed *unsuccessfully*, practical programmable analog conventional systems are a step back offering major advantages. These In-the-Ear, In-the-Canal, and Behind-the-Ear instruments have been available since 1988 with market sales accounting for 2.5 percent of sales in 1992. Programmable hearing aids may be adjusted precisely to the particular hearing impairment. The instrument may be reprogrammed at any time in a relaxed dialogue-oriented atmosphere. Direct comparisons of different setting variations help optimize speech comprehension. This type of hearing aid is ideal for fluctuating hearing thresholds often associated with Meniere's Disease.

*Body Hearing Aids:* This long-time staple of amplification use presently accounts for less than one percent of the market. The reduction in use of this model of hearing aid is primarily a result of the increased flexibility and power of smaller instruments. Of special interest in this form of amplification is frequency transposition. The basis for this circuitry is transposition of speech frequencies downward to take advantage of low frequency "corners" of hearing or to map frequencies upward to what might be termed "ultra-audiometric" frequen-



Figure 5. In-the-ear hearing aid.

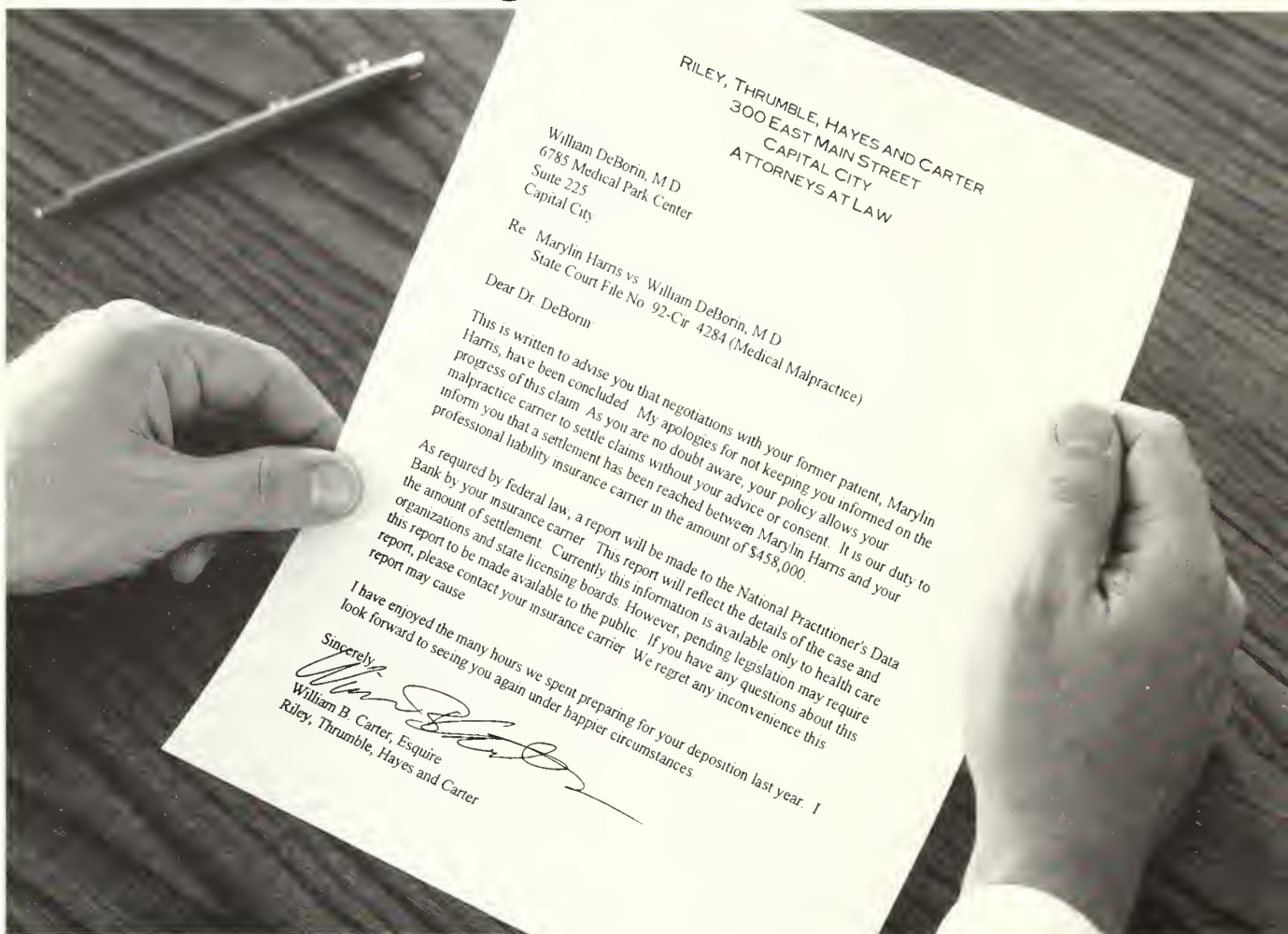
cy range above eight kilohertz. Such units may be appropriate for profoundly hearing impaired users who are possible candidates for cochlear implants or vibrotactile systems. Sound sensations are unlike those provided by a conventional hearing aid.

The only implantable device for conductive hearing loss was marketed in 1986 for a specific, yet small group of patients. Patients for whom the implantable device is indicated are those with a conductive hearing loss which cannot be corrected surgically and who cannot wear a conventional hearing aid. The reasons for inability to wear a hearing aid may be due to chronic drainage from the ear which precludes use of an In-the-Ear aid due to absence or stenosis of the external ear canal from congenital defect or acquired deformity.

Various amplification arrangements involving BTEs and ITEs are available in Cros and Bi-Cros units for unilateral or bilateral hearing loss with only one aidable ear.

The first positive step in remediation of hearing loss should be taken by the physician. Understanding of your patient's needs and a realistic expectation of what can be accomplished with hearing aids will lead to correct diagnosis and referral as appropriate. □

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# COCHLEAR IMPLANTS

TIMOTHY COURVILLE, M. D.\*

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Since 1957, when the use of an "electronic cochlea" to stimulate the auditory nerve was first reported, the management of profoundly deaf patients has permanently changed. All devices, past and present, have similar functions and components. Essentially, the devices replace the cochlear hair cell system by converting mechanical sound energy into an electrical stimulus which is delivered directly to the cochlear nerve. The components include a microphone which sends acoustic information to a processor which translates the acoustic input into a digital electrical signal that is delivered to an implanted electrode near the cochlear nerve. The implants consists of up to 22 independently functioning electrodes placed within the cochlea.

## SPEECH PROCESSING

All multichannel implants use different electrodes to stimulate different areas of the cochlea. The "place pitch" theory states that basal cochlear stimulation elicits perception of higher frequency (pitch) sound, while stimulation of the cochlear membranes near the apex is interpreted by the central nervous system as low pitch.

The speech processor converts the acoustic signal from the microphone into a digital signal. The processor then extracts key features of speech from this signal. The fundamental frequency of the speech being processed is then presented to the cochlea at the corresponding electrode "pitch place." The first and second format frequencies (overtones), and three higher frequency bands are also extracted from the digital signal and delivered to the cochlea. These formats are important for vowel and speech recognition. The amplitude of these signals determines the perceived

loudness of the stimuli. These inputs are "tunable" by the audiologist after implantation and frequently adjusted depending on the patient's threshold and comfort level.

## IMPLANT CANDIDATES

The present FDA guidelines for cochlear implantation in adults include all of the following: (1) Profound bilateral sensorineural hearing loss (nerve deafness greater than 95 db), (2) No benefit from conventional hearing aids, and (3) appropriate motivation in postlingually deafened patients. In addition to these criteria, the development of speech and language skills prior to hearing loss have remained a prerequisite in adult patients. Prelinguistically deafened adults have been implanted but results have been less rewarding. Presently the criteria for adult cochlear implant candidacy is being advocated at some centers to include patients with severe (versus profound) hearing loss or those who have some benefit from hearing aids. Implant use in these patients is investigational at present but it is anticipated that cochlear implants in this group of patients will be approved.

The guidelines for cochlear implantation in children are similar to that of adults. The minimum age of candidacy for children is two years. Although the cochlea is adult size at birth, the surgical approach during implantation is via the mastoid antrum and facial recess which are not adequately developed until two years of age. Adequate evaluation with hearing aides, tactile devices and/or frequency transposition devices should be utilized to determine functional level.

## PRE-OPERATIVE EVALUATION

A thorough otologic history is taken to determine the etiology of hearing loss. Often hearing loss can be attributed to temporal bone trauma, exposure to ototoxic drugs or infec-

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tious causes. Meningitis and labyrinthitis may cause early childhood deafness, but many causes may not be identified. Regardless of the etiology, as long as neural elements of the auditory system are present, implantation may be considered.

A thorough physical examination of the head and neck is performed. Attention is directed to the position of the auricle and its shape. Auricular anomalies often indicate other congenital anomalies of the middle ear, mastoid bone and facial nerve. Special consideration must be given to patients with congenital syndromes involving cochlear development.

The tympanic membrane must also be intact and free of disease. If otitis media is present or a recent history of chronic otitis is obtained, these conditions must be either medically or surgically corrected prior to implantation. Previous mastoid surgery is not necessarily a contraindication to implantation.

Evaluation includes a CT scan of the temporal bones. This allows evaluation of the cochlear duct which can become obliterated by fibrous tissue or bone after labyrinthitis. Although these patients are still implant candidates, the length of electrode array that can be placed into the cochlear may be limited. Patients with the Michel deformity lack development of the cochlea, while patients with the Mondini deformity are disposed to perilymphatic leaks. Although these anomalies may preclude full electrode insertion, as few as eight electrodes can result in functional improvement.

Audiologic evaluation is performed both with and without high power hearing aides. This evaluation includes pure tone testing, speech perception and discrimination, lipreading and functional communication assessments. Electrical promontory stimulation is performed to ensure a hearing perception in the ear being considered.

Patients may undergo psychological testing to identify those suffering from organic brain disease, mental illness, or mental retardation. Speech and language testing may also be con-

sidered. Extensive review of realistic expectations of the device are discussed.

## SURGERY

The initial incision used at the Medical University of South Carolina is an interiorly based post-auricular flap. The incision is made full thickness through the scalp onto the pericranium. The flap is then elevated leaving the pericranium down in adults. In children the pericranium is elevated with the scalp to maintain adequate soft tissue coverage. After the flap is elevated a limited mastoidectomy is performed exposing the short process of the incus. With this landmark in view, the middle ear space is entered anterior to the facial nerve and posterior to the tympanic membrane (facial recess). The round window niche is visualized through this opening inferior to the stapes. A small hole is drilled anterior-inferior to the round window niche into the scala tympani of the cochlea. The electrode is then inserted through this opening and until the 22 active electrodes are within the cochlea. The round window area is sealed with small pieces of fascia. The receiver package is then recessed into the temporal cranium and secured. The skin flaps are sutured in layers and a drain is placed in the scalp wound. The drain is removed on post-operative day one and the patient is discharged with a pressure bandage overlying the wound. The implant is activated six to eight weeks post-operatively. Adjustments to the processor occur over several months. Ongoing auditory processing improvements continue to be observed for 36 months or more.

## MEDICAL UNIVERSITY OF SOUTH CAROLINA RESULTS

Fifteen patients have received cochlear implants at the Medical University of South Carolina since 1991. In our series there have been no operative complications. The most common reported complication in the literature is breakdown of the scalp flap. This may be prevented by preserving the post auricular artery within the design of the flap. Uncom-


mon complications include infection and facial nerve injury.

All patients who have received cochlear implants have been activated and use their devices daily. Individual performance varies but all patients have documented improved vocal monitoring, perception of environmental sounds and improvement in functional communication. A typical cochlear implant audiogram shows thresholds at 30db (mild loss range) for frequencies 250 Hz to 6000 Hz. (speech frequencies). Typical auditory performance (no visual cues) for speech discrimination increased from an average of two percent to 71 percent with the best being 88 percent. When auditory and visual cues (lipreading) are available speech discrimination improved to an average of 91 percent, with the best being 100 percent. One of our implant users can converse normally on the telephone.

When either the patients or their families were asked if the implant improved communi-

cations skills, all recipients and their families report improved lipreading and communication skills. When asked if they would repeat the procedure, all recipients responded positively.

## CONCLUSION

Cochlear implantation can provide profoundly deaf adults and children a means to improve their lipreading and communication skills. The procedure has a low complication rate, is well tolerated by patients, and should be considered in all motivated patients with severe to profound sensorineural hearing loss. 

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# SNORING AND SLEEP APNEA: EVALUATION AND TREATMENT

JOHN G. PHILLIPS, M. D.\*

CHARLES E. SMITH, M. D., D. M. D.\*

Sleep-related problems are one of the more common complaints presenting to the otolaryngologists' office. Improved patient awareness and a new appreciation by the physician for the importance of sound sleep has brought standardized evaluation and treatment for sleep disorders into the mainstream of medical thinking.

Generally the patient comes to the office at the urging of his spouse after years of worsening snoring. Not uncommonly the spouse states that not only has the snoring worsened, but that now the patient (usually male) appears to stop breathing for several seconds many times during the night. At this point, the physician should consider that the patient may have Obstructive Sleep Apnea Syndrome (OSAS) and a thorough history and physical, as well as objective medical tests, may be required. Some definitions needed for discussion of OSAS are reviewed in Table 1.<sup>1</sup>

## EVALUATION

The two cardinal manifestations of OSAS in adults are loud snoring and daytime sleepiness, and any person having these should be considered to have sleep apnea until proven otherwise. Not only can OSAS lead to poor sleep, daytime somnolence and behavioral changes, it can also cause hypoxia, hypercapnia and acidosis, which may lead to systemic or pulmonary hypertension, cor pulmonale, cardiac dysrhythmias, nocturnal angina and, even death. Some minor clinical features of OSAS not seen in every patient are enumerated in Table 2.<sup>2</sup>

Physical features commonly found in patients with OSAS include obesity, nasal obstruction, hypertrophied tonsils and adenoids, redundant

TABLE 1

**APNEA** – Cessation of airflow at the nostrils and the mouth for at least 10 seconds.

**HYPOPNEA** – Fall in average tidal volume by more than 50 percent.

**RESPIRATORY DISTURBANCE INDEX** –  
Apnea and hypopneas per hour of sleep

**SLEEP APNEA SYNDROME** – Apnea Index equals or exceeds five episodes per hour or 30 over seven hours. The syndrome may be central, obstructive or mixed in nature.

pharyngeal mucosa and micrognathia.

Diagnostic testing centers on the polysomnogram, and consists of simultaneous measurements during sleep of electronystagmogram (ENG), electroencephalogram (EEG), electrocardiogram (EKG), chest respiration, nasal and oral airflow, oxygen saturation and myohyoid muscle tone, as well as sleep latency, which measures the time it takes a subject to fall asleep. The study has traditionally been performed in a sleep lab. However, the use of in-home studies has become more common and is usually substantially less expensive. The severity of OSAS can be gauged by the findings on the polysomnogram. Generally a respiratory disturbance index of greater or equal to 20 events per hour and an oxygen desaturation to below 90 percent are significant and may require treatment.

Other tests for OSAS that may be performed include fiberoptic endoscopy to localize the site of obstruction, cephalometric x-rays, computerized tomography (CT) scans or fluoroscopy of the upper airway and pulmonary function tests.

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**TABLE 2**  
**CLINICAL MANIFESTATIONS OF**  
**OBSTRUCTIVE SLEEP APNEA SYNDROME IN**  
**THE ADULT**

<b>MAJOR:</b>	Snoring Day time sleepiness
<b>MINOR:</b>	Physically restless sleep Personality change Cognitive impairment Headaches Morning confusion Nocturia Nocturnal enuresis Impotence

### TREATMENT

When the diagnosis of OSAS has been made and the severity assessed, treatment modalities must be considered. Medical treatment of OSAS should be the initial therapy considered in most cases. Since obesity is a common finding in patients with OSAS, weight loss will generally improve the severity of the sleep apnea.<sup>3</sup> The mainstay of medical treatment is continuous positive airway pressure (CPAP).<sup>4</sup> This has produced response rates as high as 95 percent, although compliance is a problem in a significant number of patients, given the noise and necessity of wearing a mask every night. CPAP works by stenting open the hypotonic and collapsible pharyngeal and hypopharyngeal tissues; thus providing an open airway. More recently various oral prosthetic devices have proven effective in some patients. The devices function by keeping the tongue thrust forward during sleep. Again, compliance has been a problem.

### SURGICAL MANAGEMENT

Surgical procedures have been developed for the treatment of OSAS as well as snoring. Nasal obstruction, although previously dismissed as a cause of OSAS and snoring, can be a major factor in the disorder because the nasal airway is the preferred route of respiration both during sleep and when awake. It is suggested that the nasal obstruction should be

treated if symptomatic and is often done in conjunction with other surgical options. Nasal obstruction can be secondary to adenoid hyperplasia, mucosal edema, turbinate hypertrophy, polyps, nasopharyngeal stenosis or a deviated nasal septum.

Since the site of obstruction in patients with OSAS is often within the pharynx, the focus of the most commonly-performed surgical procedure is to remove the redundant tissue found in that area. The uvulopalatopharyngoplasty (UPPP), as originally described by Fujita et al (1981)<sup>5</sup> or one of its many modifications, effectively opens the upper airway by debulking the palate and the lateral pharyngeal walls, diminishing collapse of the tissues into the airway space. It has been about 50 to 60 percent successful in correction of OSAS and about 95 percent successful in relieving snoring. Indications for UPPP, as opposed to nasal CPAP, would include failure to comply with the CPAP, and the non-obese, younger adult not wishing to wear a CPAP mask.

Tracheotomy is a procedure which is reserved for life-threatening cases of OSAS which have not responded to CPAP and in which UPPP is unlikely to provide significant improvement in apnea index. Although the procedure is not desirable to the patient, it may provide a temporary solution to the apnea while the patient is on a weight-loss program or recovering from a severe complication of OSAS.

Other less often performed surgical procedures include mandibular advancement, hyoid suspension and base of tongue resection. Also, a relatively new technique in the treatment of snoring is CO<sub>2</sub> laser (LAUP).<sup>6</sup> Performed as an outpatient office procedure in two to five visits under local anesthesia, the soft palate and uvula are reduced in size; thus reducing the vibrating surface which causes snoring. LAUP is indicated for snoring, but not for OSAS. Cure rates of 70 to 77 percent have been reported with little morbidity.

In conclusion, sleep apnea and snoring is a widespread disorder and has significant social and medical consequences. Awareness of the



consequences and being able to recognize the problem should significantly lower the long-term morbidity associated with the disorder. □

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# HAY FEVER: PHARMACOTHERAPY OR IMMUNOTHERAPY?

WILLIAM J. FRAVEL, M. D.\*

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Hay fever is a common condition which affects between 30 and 50 million Americans each year with significant symptoms occurring over an average of 19 weeks annually. The condition typically occurs early in life. In one survey, 31.5 percent of the patients reported the onset of symptoms before the age of 10. Temporary disability from hay fever results in one to two million days lost from school and 3.5 million work days lost annually. This adds up to an annual cost of greater than \$700 million in lost days and medical expenses.<sup>1,2</sup>

The spectrum of symptoms includes rhinitis, sneezing, itchy, swollen and watery eyes, post-nasal drainage, sore throat, fullness in the ears, fatigue and headache. Intensity of symptoms ranges from mild to severe, and frequency varies from seasonal to perennial. With mild symptoms, the patient may only require antihistamines on an as needed basis. Moderate to severe symptoms, especially over a long season, trigger frequent visits to the physician's office.

Chronic hay fever, if ignored or inadequately treated, may lead to complications that can be moderately disabling. Misuse of over-the-counter nasal sprays results in chronic rhinitis medicamentosa. In the average ENT office, we frequently see patients whose serous otitis media has progressed to repeated ventilation tubes, atelectatic ears, and conductive hearing loss. Patients with chronic nasal inflammation from allergy may ultimately require extensive sinus surgery, or develop nasal polyps. This progression may be interrupted by learning to recognize which hay fever patient is at greater risk and by giving that patient appropriate, aggressive and cost-effective treatment.

What is the best course of action for these patients who frequently present to our offices? When is pharmacotherapy appropriate? What are some guidelines to determine which patients should be placed on immunotherapy?

## CLINICAL APPROACH

The successful management of inhalant allergy is based on the triad of avoidance of the offending allergen, and pharmacotherapy and/or immunotherapy as needed.

## AVOIDANCE

Regardless of what else is done, it is important not to ignore this phase of the treatment. The empirical environmental control of dust, molds, animal dander, smoke and other chemical odors and fumes within the home may be enough to spare some patients extensive pharmacotherapy or immunotherapy. Treating carpets and drapes for mites, enclosing mattresses and pillows in vinyl cases, eliminating mold in showers and air conditioning ducts, and installing a high-efficiency air filter are some common "tools" of the avoidance approach. Cost-effective allergy "screening tests" (either skin tests or in vitro tests) may help pinpoint the offending allergens (e.g., dust mite, cat, common molds) which need to be eliminated. The expense of a complete allergy work-up is not necessary to obtain useful information about the general areas that are affecting the patient. Using the patient's history as a guide, a cost-effective screening panel of perhaps six to 10 antigens accomplishes this purpose. Drugs that cause nasal congestion (e.g., some antihypertensive drugs and antidepressants) may be replaced and any foods to which the patient is allergic should be eliminated from the diet.<sup>3</sup> For the patient with mild to moderate symp-

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toms occurring for three to four weeks one or two seasons each year, avoidance and/or pharmacotherapy are usually sufficient.

### PHARMACOTHERAPY

Unfortunately many troublesome aeroallergens cannot be avoided. Hypersensitivity to such allergens is one of the major indications for pharmacotherapy or immunotherapy.<sup>3</sup> With the availability of H<sub>1</sub> receptor antagonist antihistamines, including the new non-sedating varieties, cromolyn sodium, and topical and oral corticosteroids, a combination (supplemented by decongestants as needed) can usually be found that will maximize relief for the majority of patients. However, there are many potential adverse reactions to and drug interactions with both antihistamines and decongestants. Therefore, in some patients immunotherapy in combination with avoidance is recommended.

### IMMUNOTHERAPY

Immunotherapy is usually not required in the management of patients with single season or mild two-season hay fever. However, it does offer the only chance for "cure" for inhalant sensitivities.<sup>3</sup> Controlled studies establish the effectiveness of immunotherapy for allergic rhinitis related to various antigens.<sup>4</sup> Unlike pharmacotherapy, immunotherapy produces various changes in the immune system which may be responsible for the relief of allergic symptoms. These are: (1) rise of serum IgG "blocking antibodies," (2) suppression in the seasonal rise of IgE antibodies, (3) a decrease of specific IgE antibodies, (4) an increase of blocking IgA and IgG antibodies in secretions, (5) reduced basophil reactivity to allergens and (6) reduced in vitro lymphocyte responsiveness to allergens.<sup>4</sup>

When used properly, immunotherapy can be cost effective. If it prevents repeated office visits, sinus surgery, lost days at work, and significantly decreases or eliminates the medication required to control symptoms, immunotherapy can be justified, as the daily use of one of the new non-sedating antihis-

tamines and a topical steroid is often more expensive than immunotherapy. Also, if quantitative testing is used as a guide to the initial dosing, strong therapeutic maintenance doses can be reached sooner and as safely as by the single dilution testing approach.<sup>5</sup>

The following table modified from Mabry<sup>3</sup> may serve as a useful guide for initiating immunotherapy.

**TABLE 1. GUIDELINES FOR IMMUNOTHERAPY**

Consider immunotherapy when one or more of the following is true:

1. Symptoms are not controlled by pharmacotherapy.
2. Pharmacotherapy is contraindicated due to adverse reactions or drug interactions.
3. The allergen is not easily avoidable.
4. Symptoms are perennial or severe.
5. Complications (e.g., chronic sinusitis, nasal polyps, chronic serous otitis media) occur.

### SUMMARY

The total management of the patient with hay fever involves a multifaceted approach including environmental control, pharmacotherapy, and immunotherapy. Pharmacotherapy and immunotherapy are complementary methods which the allergist may utilize as appropriate to each individual patient's needs. □

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# IF SURGERY IS THE LAST OPTION FOR SINUSITIS, WHAT IS THE FIRST?

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In the United States, sinusitis affects almost all of us at one time or another during our lifetime. Fifty million patients<sup>1</sup> during the next calendar year will have treatment for a sinus infection, and treatment cost will exceed a billion dollars. Even then, chronic sinusitis maybe the most under diagnosed disease in this country.

There are four sets of sinuses, i. e.; the maxillary, ethmoid, sphenoid and frontal sinuses.<sup>2</sup> Pneumatization of the sinuses continues well into adulthood. This is one of the reasons why many patients may not develop significant problems until later in life when further growth causes anatomic abnormalities. The function of the sinuses include humidification and secretion of up to a quart of mucous a day to help cleanse the air that is inhaled. If the sinuses were solid bone, the head would weigh substantially more and the voice would be substantially less resonant.

Over the last dozen years a much better understanding of what causes sinusitis has evolved with the concept of the osteomeatal complex.<sup>3</sup> This is the region in the nose where the anterior ethmoid, maxillary and frontal sinuses all ventilate. Blockage in this area appears to be the key component in the cause of chronic sinusitis. There are many contributing factors to obstruction of the osteomeatal unit including environmental (smoking, air temperature, or chemicals), physiological (vasomotor, hormonal, ciliary, dyskinesia, cystic fibrosis, and dehydration), or mechanical (septal deviation, excess pneumatization of the sinuses, foreign body, polyps). Once obstruction occurs, oxygen levels decrease then secondary infection develops, and the

resulting symptoms include pain, pressure, headaches and post nasal drainage which may be clear at first but may become purulent and occasionally bloody. A precipitating upper respiratory tract infection commonly initiates the obstruction with allergies and/or polyps being the next most frequent cause.

Our goals are to diagnosis whether infection is present and prescribe appropriate treatment. Often in the initial stage it is to difficult to discern whether the patient is having an allergic or infectious problem. Allergy symptoms include a more sudden onset of sneezing, itching eyes, with signs of inflamed conjunctiva and clear nasal drainage.<sup>4</sup> Plain x-rays may be helpful at this point but they are most diagnostic for secondary maxillary or frontal sinusitis. Unfortunately the ethmoid sinuses which are the key sinuses involved in the osteomeatal complex, are poorly visualized on plain films. Limited CT scans (the "sinus screen") can document the key area (the osteomeatal complex), adequately and are substantially less expensive than a full CT evaluation of the mid-face.

In treating chronic sinusitis our goals are reduce tissue edema, facilitate drainage and maintain patency of the sinus ostia which will prevent chronic infection from developing. The usual treatment includes antibiotics, decongestants (both topical and oral) and mucolytics.

The most common pathogens in our hospital are noted in Table 1.

Typical treatment for sinusitis in the acute phase includes a broad spectrum antibiotic that provides coverage for B-lactamase positive bacteria which are the major sinus pathogens. These antibiotics include amoxicillin-clavulanate, cefixime, loracarbef, cefaclon and cefuroxime axetil. Treatment for

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**TABLE 1**  
**Common Pathogens**  
**in Acute Maxillary Sinusitis**

<i>Bacterial species</i>	<i>Positive No. (%)**</i>
<i>Streptococcus pneumoniae</i>	92 (41%)
<i>Haemophilus influenzae</i>	79 (35%)
Anaerobes	16 (7%)
Streptococcal species	16 (17%)
<i>Moraxella catarrhalis</i>	08 (3.5%)
<i>Staphylococcus aureus</i>	07 (3%)
Other	08 (3.5%)

\*\*339 patients/383 sinus aspirates, 1975-1990.<sup>5</sup>

community acquired and uncomplicated sinusitis usually entails a 10 to 14 day course of antibiotics, with decongestants and/or mucolytics.<sup>6</sup> Topical vasoconstrictive sprays can be used if there is sufficient swelling but should be limited to three days to prevent rebound congestion. For patients with chronic or recurrent symptoms, longer treatment with antibiotics (three to four weeks) may be needed. Follow-up plain x-rays and/or nasal endoscopy can be helpful to make sure that the active process has resolved.

In the pediatric age group there are slightly different criteria. The symptomatology is the same, although it is much more common to have significant post-nasal drainage and chronic cough. The treatment course, especially for recurrent infections, should be three to four weeks. If the patient has an allergic component, then treatment of this aspect of the patients care can significantly improve the overall course of his disease process. This may include decongestants and nasal steroid sprays for the acute process and antihistamines or immunotherapy for chronic management.

For patients with acute symptoms which do not resolve in the first 72 hours with appropriate antibiotic therapy, antral irrigation frequently relieves symptoms and also allows culture. For patients with continued symptoms an extensive course of antibiotics over three or four weeks will sometimes clear the infectious process. However, adequate nasal examination should be undertaken to help eliminate

the possibility of obstructive abnormalities including anatomic (deviated septum), polyps, or tumors. If these components are present, an otolaryngology referral is in order. If after four weeks of medical therapy the patient's symptoms are not resolved, then a sinus CT screen should be obtained. In the pediatric age group the treatment course should extend for a longer period of time, at least six to eight weeks with broad spectrum antibiotics before consideration of a CT scan. The cost of CT scans is warranted in the patient who is refractory to acute treatment, or in the patient who has had a long history of recurrent infections.

When a CT scan is ordered, there will usually be one of three results. One would be evidence of continued disease which will necessitate a surgical opinion. The second result would be anatomic abnormalities such as an unusual formation of an air cell near the opening of the maxillary sinusitis, (Halle cell), or an abnormality of the middle turbinate crowding the osteomeatal complex, (concha bullosa). Finally there will be many patients who will have a normal x-ray. It is important to have the CT read by a radiologist who is both familiar with the terminology and specific abnormalities that can be present. Scans can be read as "normal" even though there are significant anatomical abnormalities. In the patients that have normal CTs or have anatomic abnormalities which appear to be minor; an allergy work-up should be considered if a patient has allergic symptoms. Often these patients will have resolution of their nasal components without surgical intervention if the allergic component is treated.

Sinus surgery obstruction of the primary sinus openings is reserved for patients who fail medical management of infection, or have major polyps, an anatomic obstruction, evidence of a tumor, foreign body, mucocoele or pyocoele.

## SUMMARY

While sinusitis effects a great many patients, the vast majority of can be controlled with medical therapy. Understanding the physiolo-



gy and aggressively treating infection will prevent many chronic problems from developing.

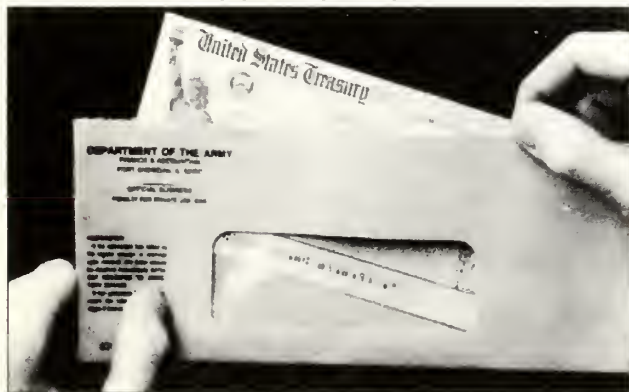


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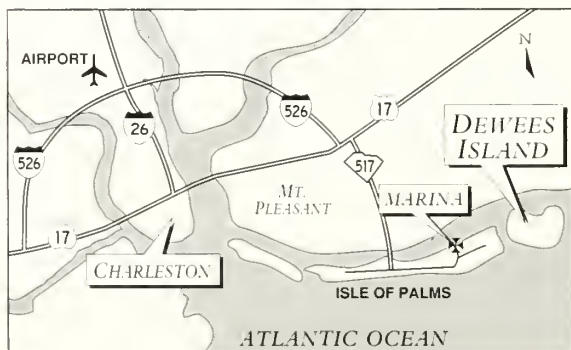
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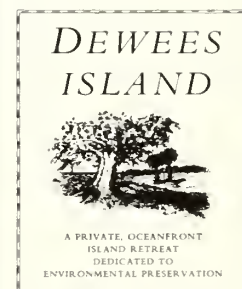
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# COMMON VOICE DISORDERS

JANE W. RIESTER, M. D.\*

MICHAEL HELLSTROM, M. D.\*\*

It is in the mechanical production of sound that the variety of conditions affecting the voice occur. Serving a dual purpose, the larynx acts to keep intruders out of the tracheobronchial tree and yet is finely tuned to produce sounds ranging from harsh and hard to soft and sweet. Most individuals quickly notice when their voice has changed and may describe hoarseness, raspy or breathy qualities. It is somewhat more difficult to describe a normal voice. In general, normal voice should have a pleasant quality, adequate pitch for gender and age, appropriate loudness and adequate flexibility for expression.<sup>1</sup>

In the next few pages, we hope to simplify the thought process involved in evaluating these patients and give a usable frame work to which to approach the patients from the non-professional, non-trained vocalist to professionals who must depend on their voice for their livelihood as well as the interesting problems posed by children, adults and the aged.

Most familiar are the conditions that change the mass of the cord. In the normal state the true vocal cords are similar to the tight strings on a musical instrument and therefore any irregularity, swelling or bowing will change the normal vibratory pattern. The cords in fact do have a mucosal wave along their length which is easily changed by any mass effect on the cord.

Edema of the vocal cords is the most commonly seen in chemical irritation of the cords by gastroesophageal reflux or tobacco abuse. Diffuse edema may also occur in hypothyroidism and as with tobacco abuse may eventually result in vocal polyps. Physical findings in patients with reflux laryngitis are consis-

tent, although not pathognomonic. Mild erythema of the pharynx is often present, but the most classic sign is arytenoid erythema, often associated with edema and may involve the entire larynx. When arytenoid erythema is caused by voice abuse and frequent throat clearing, there is usually corresponding edema at the base of the epiglottis. When the arytenoid erythema is limited to the posterior aspect of the larynx, reflux is the most common cause by far.<sup>2</sup>

These conditions may be recognizable by the history but laryngoscopy ultimately confirms the diagnosis. Indirect examination with a laryngeal mirror works well with most patients but is difficult if a strong gag reflex is present. Fiberoptic evaluation via the nares can easily be done in all patients with the use of a topical anesthetic. More recently videostrobolaryngoscopy has been used to evaluate the fine movements of the larynx and has the advantage of recording the image for comparison after treatment as well as in allowing the patient to actually see where the problem with the voice lies. This new technique allows the physician to evaluate the asymmetry of the movement of the vocal folds, glottic closure, amplitude of the vocal folds and the mucosal wave form.

Because of the vast number of diseases that produce a vocal change in children, some of which are life-threatening, progressive change not reverting to normal for a suitable length of time should stimulate a thorough exam.<sup>3</sup> Laryngomalacia, webs of the glottic area, subglottic stenosis, paralysis of the vocal cords, posterior clefts and laryngotracheoesophageal clefts are associated with voice change. Papillomas of the larynx require early diagnosis and treatment for long term therapeutic results.

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Vocal nodules are commonly seen in both children and adults from over use or abuse of the voice. Much like the callus on the skin, the vocal cords will form a firm nodule from the repetitive beating together of the cords free edge. Once the pattern of the abuse is stopped they may resolve spontaneously though allowing a speech therapist to educate patients regarding proper voice use and avoidance of bad vocal habits will not only speed the recovery process but hopefully prevent another recurrence in the future. The elimination of any other irritants such as tobacco and acid reflux is also helpful.

Hoarseness accompanied by breathiness, dysphagia, vocal fatigue and aspiration is commonly seen with the paralyzed cord. Trauma is the leading cause of this injury and may be iatrogenic (surgery, intubation) or result from external injury or pressure on the recurrent laryngeal nerve. A smaller percentage of vocal paralysis occurs from viral injury and may resolve over a period of months. The injection of Teflon paste into the affected cord has long been a means of "medializing" the cord to allow for adequate glottic closure during phonation and swallowing. The disadvantage of this technique is that it alters the mass of the cord, making it difficult to attain the pre-injury level of performance. In addition the paste cannot be removed once placed and results in an irreversible change to the cord structure. Since the mid-1970s, Isshiki type I thyroplasty has been gaining popularity for treatment of the paralyzed cord as it not only avoids changing the vocal mass but is also reversible. Under local anesthesia a "window" of laryngeal cartilage overlying the paralyzed cord is removed and a silastic block is fashioned so as to push the cord to the midline once the block is secured in place.<sup>4</sup> The use of a fiberoptic scope allows visualization of the larynx once in position and can be assessed while the patient vocalizes.

Adductor spasmodic dysphonia vocal disorders occurring in the absence of any structural

or movement abnormalities in the past were termed functional.<sup>5</sup> This disorder is characterized by uncontrolled pitch breaks and hoarseness with a loss of voice. Patients in large series of study have often been through speech therapy, psychological testing and some psychiatric therapy. A relatively new treatment for this disorder involves injection of Botulinum toxin. The therapeutic effects of the injection last approximately six months and patients rarely return to the pre-injection state. Side effects of injections include reduction in swallowing speed and voice volume.<sup>6</sup>

Not to be overlooked is the patient with a significant history of tobacco abuse, who is seen by the family doctor with odynophagia, otalgia or hoarseness. If these patients do not improve within a week or two, they should be evaluated by the otolaryngologist. Early carcinomas of the vocal cords can be present with any of these symptoms and are curable in over 90 percent of patients if found at an early stage.

In summary, vocal changes usually represent fairly common entities but may signal life threatening diseases. New techniques such as stroboscopy for evaluation of the vocal cords and Botulinum toxin for spasmodic dysphonia are recent introductions that can vastly improve the treatment and diagnosis of vocal abnormalities. □

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# OPTIONS IN COSMETIC REHABILITATION OF THE AGING FACE

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## EVOLUTION OF THE "AGING FACE"

The sequence of changes that occur in the aging face is relatively uniform, but the rate may vary from person to person.<sup>1</sup> Beginning at about age 25, the eyebrows begin to descend from a point well above the supraorbital bony rim to a point well below. Sagging of facial skin first becomes apparent at about age 30, particularly where the thin skin of the upper eyelids overlays the normal palpebral creases. At about the same time the melolabial (nasolabial) folds begin to deepen. Forehead wrinkles and horizontal skin lines at the lateral canthus begin to appear at about 40 years of age, and sagging of the mandibular line occurs producing the undesirable "jowled" appearance. The next decade introduces further changes about the eyes, with downward slanting of the lateral canthus. The nasal tip starts to descend, and wrinkles appear in the perioral area and neck. Some resorption of adipose tissue in the temporal and cheek area occurs, which gives the face a hollowed appearance. At 60 years of age, the illusion of decreased eye size becomes pronounced and the skin is thinner. By 70 years, all these changes combine with progressive subcutaneous fat resorption.

Other changes take place in specific areas. The bones of the skull become thinner, thus causing the entire skull to become smaller. This further aggravates the problem of excess overlying facial tissue. Eyebrow descent creates excess skin above the eyes which, combined with a weakening of the orbital septum, allows intraorbital fat to herniate and create palpebral bags. Resorption of alveolar bone

results in an excess of perioral soft tissue. The well defined angle between the submandibular line and the neck is lost with age. The anterior free edges of the platysma separate and lose tone, which creates the anterior banding or "turkey gobbler" deformity so characteristic of the aging neck. Frequently a deposit of fat is found in the submental area.

The facial skin itself ages both intrinsically and extrinsically.<sup>2</sup> Intrinsic aging refers to the effects of chronology, and consists of atrophy and loss of ground substance. Extrinsic aging manifests as dysplasia and alteration of structure. These changes result from environmental factors such as ultraviolet radiation, cigarette smoke,<sup>3</sup> alcohol and other toxins. Along with wrinkling, other age-related changes in skin include a loss of elasticity, increased laxity, roughness, sallowness, telangiectasia and mottled pigmentation.

## PSYCHOLOGY AND ATTITUDES TOWARDS AESTHETIC AND AGE REHABILITATION SURGERY

In the past 20 years, there has been an extraordinary increase in surgery for appearance. Over 10 years ago, *Newsweek* described facelifts as the "newest middle class status symbol."<sup>4</sup> People from all walks of life seek cosmetic surgery for various reasons. The majority of today's aesthetic surgery patients are not wealthy, but have middle to upper class incomes.

Not only are more people seeking such surgery, but the surgery is now available to a wider group. Fees for aesthetic surgery have not risen as rapidly as those for many other types of medical care, which has made the surgery more affordable. Explosive development of outpatient ambulatory surgery centers

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and office-based surgery as well as improvements in anesthetic safety have led to rapid advancement and improvement in surgical techniques. More patients are obtaining better results.

Concern for one's appearance (as opposed to a lack thereof) is correlated with healthy self-esteem. The patient who presents with facial signs of aging may have been entirely satisfied with his or her appearance in the past, but may now be dissatisfied simply as a result of the changes brought about by aging. Aesthetic surgery is a means of helping an individual improve his or her self-image. Significant improvements in self-esteem after facelift surgery are evident time and time again in formal research and in clinical practice.

As a person ages, his or her body image lags behind actual physical changes. We tend to perceive aging in others before perceiving it in ourselves. (Remember your last class reunion?) Facial rejuvenation surgery brings one's appearance more in line with one's self-image. While rejuvenating facial surgery obviously has limitations, the obvious advantages for career and social aspects of life are undeniable. Up until a few years ago even the IRS allowed cosmetic surgery as a business deduction for professional people!

### **OPTIONS THE FACIAL PLASTIC SURGEON CAN OFFER FOR COSMETIC REHABILITATION OF THE AGING FACE<sup>5</sup>**

*Facelift* (Rhytidectomy) is generally divided into three components: the cheek lift, the neck lift and, the brow, or forehead lift (discussed below). It is often desirable to include submental liposuction to correct a "double chin." Local anesthesia with or without I.V. sedation or, if preferred, general anesthesia is used. Incisions are well concealed around the ear and in hairbearing areas of the scalp to camouflage the scars. The superficial musculoaponeurotic system (SMAS, a connective tissue layer continuous (or contiguous) inferiorly with the platysma) is imbricated and excess skin is excised. A firm dressing is

worn for one or more days. Swelling and discomfort, usually not painful, will generally subside in seven to 10 days, and the patient is usually quite presentable in about 10 days.

Improvement depends on the amount of laxity of the skin and underlying SMAS. If laxity is marked, results may be dramatic. The aim of the aesthetic surgeon is to create a natural, rested appearance. Improvement usually lasts seven to 10 years with gradual laxity occurring as time progresses. Facelifts turn back the hands of time but do not stop the clock from running. The risks with an experienced surgeon's hands are minimal.

*Forehead and Brow Lift.* Heredity, age, skin abuse and sun exposure can cause the brow, temples and forehead to relax and descend. The result can be a fatigued or sad look and, when pronounced, even an angry look. The forehead lift, often a component of the facelift, is designed to elevate the brows and diminish forehead wrinkles through excision of skin in or behind the hairline, completely hiding the scar. Frown lines, due to hyperactive procerus or corrugator supercilii muscles, are treated with cautery or excision.

The brow lift is designed to remove skin just above the brow to improve drooping of the brows themselves. The direct brow lift with the incision just at the upper border of the hairy brow is sometimes effective in women, while the mid-forehead brow lift is most successful in men since they usually have numerous horizontal rhytids to hide the scars.

*Blepharoplasty* is performed on both the upper and lower eyelids to remove excess skin (dermatochalasis) and orbital fat herniating through the orbital septum. Incisions are made in natural skin folds, making them virtually unnoticeable after they are healed. In patients where there is minimal excess skin overlying herniated fat pads of the lower lid, a transconjunctival approach to the fat can be used. This approach avoids any skin incision whatsoever, and promotes somewhat quicker healing.

*Chemical Peel* (Chemexfoliation) is used to smooth out fine wrinkles in areas where surgery is less effective, and is often done in



conjunction with other facial plastic surgery. Over the years, fine lines known as "crows feet" appear around the eyes, and small vertical crinkles develop on the lips. Some patients develop rows of fine wrinkles across their cheeks, giving them a weathered appearance. A facelift can remove sags or bulges, but a chemical peel is required to smooth out the fine wrinkles.

The agents and techniques used in chemical peeling are undergoing evolution.<sup>6</sup> Presently, several options include the deep peel (i.e., Baker's phenol solution for penetration to midreticular dermis), the medium peel (i.e., 35 to 50 percent TCA for penetration to upper reticular dermis) and the superficial peel (i.e., 10 to 25 percent TCA or Jessner's solution for penetration to papillary dermis). The newer, milder, glycolic acid peel is ideal for younger patients and can be repeated. Local anesthesia with IV sedation is generally used with the deeper peels. Risk includes scarring, which is very rare, and inappropriate pigmentation problems which are rare if sun exposure is avoided.

*Surgical Treatment of Hair Loss.* Nearly two-thirds of the male population and some women suffer some hair loss with aging. The most simple and widely used technique for hair restoration is called a punch graft. Small "plugs" of scalp and hair follicles (punch grafts) are used to replenish bald areas of the scalp. Because of the need for spacing to allow revascularization of the grafts, it takes four or five sessions to complete an area. Sessions can be as frequent as three weeks apart. About six weeks after transplant, the hair falls out of the grafts, with new hair appearing in about three months. The "doll's hair" appearance which hampered earlier transplant efforts has been dramatically reduced due to newer techniques using mini and micro plugs (one to two hairs per graft) to feather hairlines, along with proper styling.

Another approach to hair restoration by using scalp flaps, requires more surgery but affords better results. There are several variations but, in all of them, large sections of hair-

bearing scalp are elevated at one end and rotated from the temporal and occipital scalp to the bald frontal area. Because the hair is never separated from its blood supply, there is no temporary hair loss. Coverage is more rapid and is permanent. A uniform hair density is achieved, which makes it easier to style the hair in a conventional manner. The possibility of partial loss of the flap because of vascular embarrassment is a consideration.

*Rhinoplasty.* Though commonly thought of as a procedure for the young and middle-aged, rhinoplasty has gained increased popularity in the older population as well. Significant changes associated with the aging nose are due to gravity and atrophy along with continuous life-long nasal growth. Techniques used in the younger population are modified significantly when applied in the aging patient.

*Facial implants* play an increasingly significant part in surgical improvement of the aging face. Various alloplastic implants of firm polymers, and graft materials such as Gortex<sup>®</sup> can be used to fill in areas where atrophy of subcutaneous fat has begun to show the effects of aging. Other smaller defects can be corrected temporarily (three to five months) with the implantation of commercially prepared collagen.

The Figure represents a case example of a patient who underwent facelift, forehead lift, blepharoplasty and perioral chemical peel.

## AN APPEAL TO THE PRIMARY CARE SPECIALIST

When should you consider referring a patient for facial rejuvenation surgery? Proper evaluation of prospective patients requires surgical plus psychological skill and experience.<sup>7</sup> The patient's motivation should be self generated and not the result of external forces. The patient should have a healthy self-image. Finally, the patient should have reasonable expectations.

Patients who are widowed or divorced must be free of major depression so that they can withstand the brief emotional setback which sometimes occurs during the short postopera-



**Figure.** Preoperative (left) and postoperative (right) photograph of a patient who underwent facelift, forehead lift and blepharoplasty.

tive recovery period. Nonetheless, these patients can often benefit tremendously from increased self-esteem after their emotional trauma.

In terms of preventive medicine, continue to warn all "tan" patients about the effects of sun on aging. As a means of motivating smoking cessation, what better reward could a patient have upon realizing a goal of smoking abstinence than to take the money normally spent on cigarettes and use it for cosmetic rejuvenation of the aging face? □

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# Gray Matter

*"Matters of Interest  
to South Carolina  
Physicians."*

Thornton & Thorne give the medical community something to think about this month.

## LAST CALL ON DISABILITY INSURANCE

### (AT TODAY'S RATES AND CONTRACTUAL PROVISIONS)

A near crisis atmosphere exists today in the disability insurance marketplace. Insurance companies are experiencing significant losses and are scrambling to stop the bleeding. ***No class of insureds is going to be more affected by the restructuring than physicians.***

We've written several articles over the last few years predicting the changes that are occurring now. We advised physicians to buy all the noncancelable, individual disability insurance they could before the changes occurred. Time to act is running short.

In this space in January, 1994, we wrote ***"...many changes have occurred in the 90's and many more will occur in the next few years. The products available to protect your income in January 1996 may bear little resemblance to those available today"***.

Reviewing these comments in October of 1994, we now see that we were optimistic. The rapidity and depth of change have gone beyond what even we expected.

**These changes have been, and will continue to be, dramatic and far-reaching.** Companies have increased premiums significantly. You can expect further increases. They will shorten benefit periods, lengthen elimination periods, restrict benefits for mental or emotional claims, and make further restrictions in the definitions in the policies.

Both health and financial underwriting will continue to get tighter. Certain medical specialties will be designated as undesirable due to adverse claims experience.



One change that has yet to happen, but will occur soon, is the introduction of *guaranteed renewable policies*. The premium for guaranteed renewable policies can be adjusted as experience dictates. The time will soon come **when you cannot purchase a policy with guaranteed premiums.**

## **GENDER BASED PRICING**

Most major companies have begun issuing policies which have sex distinct premiums. This substantially increased premiums for females. **Female physicians should purchase policies as soon as they can from a company which still uses sex distinct pricing. This opportunity will only be available for the next few months.**

## **RECOMMENDATION**

**What should you do about all of this?**  
In a 1992 article, we made the following recommendation:

***"...purchase as much individual, non-cancelable insurance as you can get. Once issued, the premium can never be changed nor any restriction put on the policy regardless of what happens in the future".***

***"You will not be healthier in the future than you are today. The premiums will not be as low as they are today. The policies will not be as good as they are today. ACT NOW."***

These comments are even more appropriate today than in 1992. SCMA members get a 25% discount on disability policies from Connecticut Mutual. This contract has not been changed since late 1992 but change is imminent. **It's the best buy available to SC physicians but you must act promptly.**

*Views expressed herein are those of the authors only and in no way represent the SCMA. We do not give tax advice. Only your attorney and accountant are qualified to do so.*



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# EVALUATION OF THE NECK MASS

JEFFREY R. FENWICK, M. D.\*

RUSSELL D. KITCH, M. D.\*

The patient presenting with a neck mass is a fairly common occurrence in the primary care setting. Evaluation of these masses requires a careful analysis of several factors in order to correctly arrive at a proper diagnostic and therapeutic approach. The inexperienced clinician may be alarmed at a large inflammatory mass presenting in a child, while initially less aggressive in evaluating a more subtle mass in an adult which more likely represents a malignancy. The differential diagnosis of the neck mass in both children and adults is quite extensive. Table 1 is a partial listing of the common etiologies of neck masses in both children and adults in the approximate order of their presenting frequencies. A detailed discussion of the various entities described in Table 1 is beyond the scope of this article. Rather, an attempt will be made to provide a practical framework for the evaluation of the neck mass as it so often presents in the primary care setting.

## HISTORY

A careful history is obviously the first place to start in evaluating a neck mass. What is the patient's age? The vast majority of all large, single neck masses presenting in children are benign and inflammatory in nature. The reverse is true in adults, with most single neck masses representing either a benign or malignant neoplasm. The clinical chronology can provide important clues. Large, rapidly appearing neck masses are often inflammatory, while slower growing masses that have been present for several weeks to months are more ominous. Are there other associated symptoms such as fever, sore throat, hoarseness, dysphagia, weight loss, night sweats, etc? Is the mass painful? Pain in a neck mass

**TABLE 1**  
**MOST COMMON ABNORMAL NECK MASSES**  
**IN CHILDREN AND ADULTS**

### CHILDREN

- palpable lymph node or mass  $\geq 1$  cm.
- 70-90 percent benign
- (a) Lymphadenitis:
  - staphylococcal, streptococcal (most common)
  - mycobacterial
  - cat scratch bacillus
  - EBV (Epstein Bar Virus)
  - CMV (Cytomegalovirus)
  - Toxoplasma
  - HIV
- (b) Congenital neck masses:
  - branchial cleft cyst
  - cystic hygroma
  - hemangioma
  - thyroglossal duct cyst
  - dermoid cyst
  - thymic cyst
- (c) Salivary Gland Infection or Obstruction
- (d) Malignancies
  - lymphoma
  - rhabdomyosarcoma
  - thyroid

### ADULTS

- palpable lymph node or mass  $\geq 1.5$  cm.
- 70-90 percent malignant
- (a) Metastatic Disease
  - squamous cell carcinoma (vast majority)
  - thyroid carcinoma
  - non-thyroid adenocarcinoma
- (b) Primary Cervical Neoplasm
  - lymphoma
  - thyroid/parathyroid
  - salivary gland
  - paragangliomas
  - vascular tumors
- (c) Congenital Neck Masses (see under children)
- (d) Lymphadenitis
- (e) Mucosal Defects
  - laryngocele
  - Zenker's Diverticulitis
- (f) Cutaneous
  - epidermal inclusion cyst
- (g) Lipoma

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of neoplastic origin is uncommon until the tumor reaches massive size. The patients social history should be noted, especially with respect to tobacco and alcohol use. Finally, the patient should be thoroughly questioned regarding previous exposure to infectious agents in other family members, as well as exposure to radiation and other environmental agents in the past.

### PHYSICAL EXAMINATION

The physical exam should include a careful assessment of the mass itself. Note should be made as to its location in the neck, its size, consistency (i. e., cystic or solid), and whether it is painful to palpation. Solid, rounded, multinodular masses which seem fixed to the underlying structures are ominous and warrant careful evaluation.

The patient presenting with a neck mass should also receive a thorough examination of rest of the head and neck in order to identify the possible source of the mass. While examination of the ears, nose, and oropharynx may seem obvious, many other potential sources can be overlooked. A mirror examination of the hypopharynx and larynx is essential and should be attempted when feasible. The scalp should be thoroughly examined for signs of infectious and neoplastic processes. A passing knowledge of the lymphatic drainage of the head and neck is helpful in identifying a source. The location of a neck mass in a specific anatomic region can direct the physician to concentrate his or her examination to those areas of the head and neck drained by that set of lymph nodes. Figure 1 shows the major anatomic areas of the neck, and the regions of the head and neck that drain into each of those areas.

### DIAGNOSTIC STUDIES

It is only after a careful history and physical examination that the physician is able to decide which diagnostic tests, if any, may be helpful in delineating the nature of a neck mass. If an infectious etiology seems likely, a CBC may be helpful. Serologies and skin tests

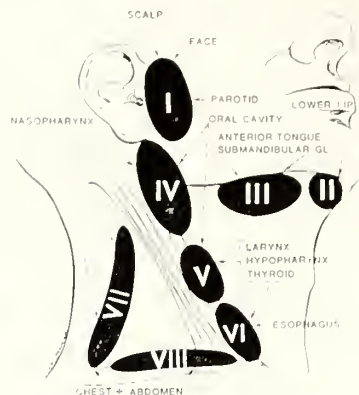


Figure 1. From *Diagnosis of Inflammatory Diseases of Cervical Lymph Nodes*, Page 17, Second edition published 1988 by Dr. Peter J. Moloy. I-Parotid; II-Submental; III-Submandibular; IV-Jugular-Digastric; V-Mid-jugular; VI-Lower jugular; VII-Spinal Accessory; VIII-Transverse cervical.

for various infectious agents such as atypical mycobacterium or cat scratch disease are of limited utility and should be obtained sparingly. A CT scan of the neck with contrast is helpful in determining the exact location and extent of the mass, as well as whether it is cystic or solid. Central necrosis and fixation to underlying structures are ominous findings. Ring enhancement can signify both infectious and malignant lesions.

MRI scanning, while helpful to the specialist, is probably less so to the primary care physician as an initial screening technique at the present time, given its cost. Ultrasound is inexpensive and quick, but in the neck its use is basically limited to ascertaining the presence of fluid in a suspected cyst or abscess cavity.

Nuclear imaging studies have their place in the evaluation of low anterior neck masses of suspected thyroid origin. Aspiration of a suspected cystic mass can provide material for both culture and cytology and can usually be done by the primary care physician. Likewise, the technique of fine needle biopsy may one day be a valuable technique for the primary care physician although at the present time its practical utility is limited by availability of the new sub-specialty of cytopathologists, usually found in large academic centers.

Finally, the use of incisional biopsy in the



primary care setting should be discouraged for the following reason. Incision into a malignant neck or salivary tumor can compromise cure, as it disrupts local lymphatics and can increase the incidences of local, regional, and distant metastases. If biopsy is contemplated, the physician must be prepared for definitive resection should frozen section study of the specimen disclose malignancy. □

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# THE EVALUATION AND DIAGNOSIS OF THE DIZZY PATIENT

W. DAVID ISENHOWER, JR., M. D.\*

RICHARD M. CARTER, M. D.

PETER C. WEBER, M. D.

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The complaint of dizziness has a plethora of implications. Dizziness is used by patients to describe weakness, lightheadedness, wooziness, a sensation of blacking out or true vertigo, or whirling sensation, where either the patient or the patient's surroundings are moving about them; even when no motion is actually present. This last description is often referred to as vertigo.

As a patient complaint, dizziness is quite common. It has been reported to be anywhere from the third to the thirteenth most frequent reason for visits to internal medicine clinics. It has also been described as one of the top five complaints seen in the emergency department. Any otolaryngologist will also tell you that dizziness or vertigo is a common office complaint.

Fortunately, most complaints of dizziness can be classified into one of five groups. The most frequent group would be that of vertigo. Vertigo may have a peripheral etiology such as Meniere's disease, benign positional vertigo, a perilymphatic fistula, an autoimmune disorder or an infectious disorder such as labyrinthitis. Central vertiginous disorders are classically those of stroke, multiple sclerosis, vascular loop syndrome or trauma. A second major category of dizziness is due to impaired perfusion. The perfusion disorder can either be of a central type such as stroke or vascular spasm or it can be more of a peripheral type such as a basilar artery insufficiency, static hypotension, an arrhythmia of some type, or it may be associated with headaches. The third

category can be described as a feeling of disequilibrium where the patient complains of an inability to walk straight or to keep one's balance when standing. These difficulties are usually associated with multiple sensory deficits. The fourth category would be those associated with metabolic disorders such as hypo- or hyperthyroidism, diabetes, syphilis or Cogan's syndrome. The last category falls under the group described as psychogenic dizziness. These types of disorders may be associated with major depression, anxiety disorders, psychosomatic type disorders and of course we also have to rule out the possibility of secondary gain such as patient seeking disability as their sole motivation for complaint of dizziness.

Fortunately, a complete history and physical examination with appropriate laboratory and electrical studies allow us to classify the patient's dizziness, pinpoint the diagnosis and offer treatment to most patients. The purpose of this paper will be to highlight some of the pertinent historical questions that need to be addressed in order to help categorize the dizziness as well as the important physical examination findings, laboratory and electrical study findings.

## HISTORY AND PHYSICAL EXAMINATION

The first priority is to establish whether or not the patient indeed has true vertigo i. e., sensation of the rooms spinning or moving about the patient or the patient spinning or moving about a stationary room. The reason for making this distinction from that of other types of dizziness, is that vertigo, as stated earlier, can usually be

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classified into peripheral or central etiologies. Vertigo also may require different studies as compared to other types of dizziness.

Patients who complain of dizziness, primarily with standing which lasts only a brief period of time, should be evaluated for orthostatic hypotension. This may be performed by taking the patient's blood pressure in the supine position and then again when standing immediately and again after five minutes. If there is a discrepancy between these blood pressures, such as a drop in blood pressure with standing, it is likely that the patient is suffering from impaired perfusion to the central nervous system with standing and thus has orthostatic hypotension. It would be important at this point to inquire what medications the patient is on, whether any new medications have been prescribed, and have the patient evaluated by his internist or cardiologist. It is also important to inquire from the patient his cardiac history especially interested in any history of myocardial infarctions, arrhythmia disorders, cardiac medications or diuretics that the patient may be currently taking, since this may also alter blood flow.

If the patient complains mainly of dizziness with head extension or at times even flexion, concern must be of basilar artery insufficiency. This diagnosis is not uncommon and is especially found in patients with cervical arthritis. A lateral and AP neck x-ray may help confirm a physician's suspicions of this diagnosis. A history of transient ischemic attacks (TIAs) should also be elicited from the patient as this can be an indication for the feeling of dizziness and may be the precipitating cause. Care should be taken to determine whether the patient is on anticoagulants at the time to help diminish the opportunity of subsequent TIAs. Regardless, patients should always be evaluated for carotid bruits. If there is also concern from the history of atherosclerosis or the patient states that there are times when he feels like he is going to pass out, further investigation with carotid Doppler studies is useful in determining the patency and the likelihood of atherosclerosis as a cause for

dizziness.

Patients whose primary complaint is that of disequilibrium need to be asked about previous strokes, history of diabetes, whether or not they have any peripheral neuropathies and also what their visual acuity is as well as if they have ever had any difficulties before with their balance nerve. Maintaining one's equilibrium is dependent upon three, but integrating somatic sensory cues. The vestibular system combined with the visual system and sensory inputs from the lower extremities aid us in maintaining balance. Removal of any one of these inputs may affect the patient's ability to maintain his or her balance, but normally one is able to compensate. Removal of two or more of these inputs may unfortunately severely alter our equilibrium. Thus in patients with balance disorders it behooves us to determine which inputs are affected in order to properly care for our patients. Thus it is important to establish whether or not the patient has any visual acuity problems, whether there is any evidence of peripheral neuropathy or any evidence of diabetes. These may all lead to difficulties in the maintenance of balance and cause disequilibrium. It is also important to inquire about previous vestibular deficiencies as well as inquiring about the use of various vestibular depressant medications.

A history of headaches may also be associated with complaints of dizziness and is not uncommon. The headaches may be of the migraine fashion or they may not. It is important to rule out any type of intracranial tumor or increase in intracranial pressure as an etiology for these headaches, but it should be noted that dizziness is a common complaint with people with headache disorders. Neurologically a seizure disorder must also be entertained as a possible etiology for a complaint of dizziness and if the patient has complaints, such as vague feelings of blacking out or headaches with the dizziness, it is imperative to have a neurological evaluation in order to rule out a seizure disorder.

The last category, psychogenic dizziness, is really a diagnosis of exclusion. It should be

considered in all patients that have either ongoing or previous psychiatric illnesses, such as major depression or anxiety disorders. It is also important to inquire about secondary gain. Some patients are filing for workers' compensation or are in litigation from post trauma and thus may have something to gain by being persistently dizzy.

Vertigo, on the other hand, requires a little bit different line of questioning. It is important to ascertain when the vertigo actually occurs. Vertigo that is associated with quick head movements or turning over in bed quickly or from looking side to side that lasts anywhere from a few seconds to approximately a few minutes and then dissipates quickly should raise the concern of benign paroxysmal positional vertigo as a diagnosis. Other etiologies could be related to cervical vertigo, especially if the patient was involved in a recent trauma. The cervical vestibular ocular reflex is well documented and this can also cause symptoms of this nature. The treatment for these two etiologies is quite different. Benign positional vertigo is treated with vestibular rehabilitation exercises whereas cervical vertigo is best treated with cervical collar and very light vestibular rehabilitation exercises. Benign positional vertigo also should have a history of being fatiguable on examination such that it is reproducible in the office setting with Hallpike's maneuvers and with repeated Hallpike's maneuvers the severity of the episode greatly diminishes. Unfortunately, Hallpike's maneuvers may fail to illicit a response thus history is still important.

Patients who complain of vertigo with straining, blowing their nose, post trauma or after a recent dive must have the consideration of a perilymphatic fistula made as an etiologic factor for their vertigo. This can best be verified with pneumatic otoscopy and also ENG pneumatic otoscopy, although neither is 100 percent diagnostic. This normally occurs from a small defect in either the round or oval window causing a leakage of perilymph especially under increased intracranial pressure.

Patients who state their dizziness is episodic

in nature, the vertiginous attacks last anywhere from one half an hour to a few hours, such that they are incapacitated and must often lie down until the attack abates, must be considered candidates of having endolymphatic hydrops or Meniere's disease. Meniere's disease is a combination of tinnitus, ear fullness, hearing loss and the vertiginous attacks that can occur episodically. These episodes maybe daily, weekly, with no real set pattern. It should also be noted that not all patients with endolymphatic hydrops experience hearing loss, tinnitus or fullness in the ears but may have some sort of combination of these.

Patients who complain of a severe attack of vertigo that lasted for approximately 24 to 48 hours and since that time are having difficulty with their balance and periodic bouts of vertigo should be questioned for an antecedent upper respiratory tract infection as this is usually associated with viral labyrinthitis.

A second major area of questioning is that of association of hearing loss. This can be well documented with an audiogram. Patients with vestibular neuronitis may or may not also have associated hearing loss which may be mild or quite severe. Patients with Meniere's disease typically demonstrate a low frequency sensorineural loss, but in those patients with long standing endolymphatic hydrops the hearing loss picture may show a typical flat sensorineural hearing loss across all frequencies. Typically, benign positional vertigo, cervical vertigo, vertigo associated with headache, does not demonstrate any correlation of hearing loss. An asymmetric sensorineural loss is important, especially if it demonstrates a discrepancy in word recognition scores as this may be a sign of an acoustic neuroma or other type of cerebellar space occupying lesion and should be investigated with magnetic resonance imaging with gadolinium.

A complete neurologic history is also important in patients with vertigo. Patients who demonstrate muscular weakness, difficulty with speech or diplopia may have a central etiology for their vertigo. Recent drug history

or medication history is important. Patients receiving certain kinds of antibiotics such as aminoglycosides, lasix, or chemotherapeutic agents may be more prone to vestibular system insults. It is also important to inquire, as stated earlier, about the overall medical condition to ensure that there is no hyper- or hypothyroidism and no history of syphilis. These metabolic factors can all be checked with routine blood screening. Cholesterol levels, thyroid function testing, hemoglobin/hematocrit (especially important in elderly patients to rule out anemia), electrolytes, glucose, calcium and magnesium are important to check.

### PHYSICAL EXAMINATION

It is important to perform a complete otolaryngologic and neurologic evaluation of the patient. This should include checking all cranial nerves, checking for symmetric muscle strength, tone and reflexes. Gait should be observed with turning both to the right and the left. A wide based gait or difficulty in being able to turn about may signal some type of central etiology. This central etiology is usually in the cerebellum. Gait may be also assessed by asking the patient to walk a straight line with his eyes closed. A patient with a cerebellum disorder will normally deviate to the affected side. Asynergia tests are also important to assess the cerebellum. The patient may stand erect with his eyes closed and is instructed to bend backwards. Normally, one will automatically flex the knees to gain postural control but in cerebellum disease no flexion occurs and the patient falls. Similarly, the patient is asked to move forward with his eyes closed. The patient with cerebellum disease may fall because the feet will advance but the trunk will not. Finally, if the patient in a supine position is asked to rise, the normal person raises the trunk, while those with cerebellum disease utilize the feet first. Testing for this dysdiadochokinesia is also important for testing cerebellar coordination on the affected side. Arm tones is yet another test. The patient stands erect, extends his arms forward; if one arm consistently drops it may be a sign of a cerebellum space lesion on that side. Bárány's

past pointing test is also useful for testing the cerebellum. The patient is asked to take his index finger and touch his nose and then touch the physician's finger repeating this movement over and over. The patient is then instructed to close his eyes and continue. Normally, no past pointing or deviation is noted, unless there is a unilateral irritative vestibular or cerebellum lesion.

Nystagmus is also assessed by asking the patient to follow the physician's index finger with only his eyes. Spontaneous nystagmus with these maneuvers is pathognomonic for some type of vestibulopathy. Upgaze nystagmus is almost always associated with a central lesion while downbeating nystagmus is pathognomonic for Arnold-Chiari malformation or basilar artery compression. The oculocelphalic reflex is also tested by asking the patient to look straight ahead and the physician passively moving the patient's head right, left, up and down. Normally, the eye should deviate in the direction opposite the movement.

### HALLPIKE'S MANEUVERS

The evaluation of positional nystagmus with patients with complaints of vertigo is also essential. These maneuvers are well known to most physicians. With the patient seated, he is asked to turn his head to the left or right very quickly as he is also quickly brought down to a supine position. The patient is then observed in for any signs of nystagmus and of vertigo. The patient is then brought back to the upright position and observed. Frontal lenses are sometimes required in order to actually visualize nystagmus. If nystagmus occurs or symptoms arise during the initial move, the test is repeated in order to check for fatiguability. Benign positional paroxysmal vertigo or BPPV is almost always associated with positional complaints that do fatigue with a nystagmus that is direction fixed. This finding almost always indicates a peripheral vestibular disorder which will usually respond to rehabilitation exercises or the maneuver of Epley. Nonfatiguability types of nystagmus or vertiginous symptoms are more worrisome



since they may indicate a central lesion. If normal Hallpike's maneuvers fail to illicit a response, it is wise to have the patient demonstrate what brings on his nystagmus in the office. It is important to remember that just because one cannot illicit a response in the office does not necessarily mean that the patient does not suffer from BPPV, it may mean that by the time the patient is seen in your office his response for that day has already fatigued.

It is essential to examine the ears thoroughly; and this is best done under the microscope. Evidence of acute otitis media, serous otitis media, cholesteatoma, tumors, or drainage from the ear may all predispose to vertigo and this condition would have to be treated first in order to determine whether or not it was the offending cause of vertigo. Pneumatic otoscopy is performed in order to determine whether or not a positive fistula test can be elicited as a possible etiology for the patient's vertigo. Examination of the nasopharynx and the nasal cavity is important to ensure no evidence of sinusitis, nasopharyngitis or nasopharyngeal mass which may be occluding the eustachian tube. Although a complete otolaryngologic examination is required, particular attention should be paid to the larynx and base of tongue if the patient complains of otalgia and then feelings of vertigo, as this may be due to referred pain from a lesion in these areas. It is important to auscultate the carotid arteries with a stethoscope in order to check for bruits. Fundoscopic examination of the eyes is important to rule out increased intracranial pressure.

### **OFFICE LABORATORY TESTING**

The first laboratory test that is obtained in the office is that of an audiogram. It is important for documentation of the patient's hearing. Many vertiginous and dizzy disorders are associated with decreased hearing. Meniere's disease, as stated earlier, is associated with a low frequency sensorineural hearing loss to a flat loss in those patients with more advanced disease. Vestibular neuronitis may show com-

plete severe sensorineural hearing loss or may demonstrate no hearing loss at all or hearing loss just in the basal turns. Asymmetric sensorineural hearing loss, especially with asymmetric decrease in word recognition scores, may be indicative of a cerebellum space occupying lesion. Fluctuating sensorineural hearing loss may be due to a perilymphatic fistula or if the fluctuating sensorineural hearing loss is bilateral, one must entertain autoimmune disease as a etiologic factor.

Complete electronystagmography or ENG testing is also essential in the evaluation of the vertiginous patient. Electrodes are placed around the eyes to measure nystagmus electrically based upon certain visual and thermal cues. Saccade movements, pendular tracking, optokinetic gaze, and positional testing are all evaluated to provide insight as to whether or not a central lesion may exist. In addition, bithermal calorics or the Torak monothermal caloric test may be utilized. Both of these tests are used to ascertain the degree of vestibular function on each side and to determine whether or not there may be a peripheral vestibular lesion. If no nystagmus is seen with cold calorics, ice water calorics are then applied in order to further test the vestibular system. Abnormal caloric testing usually indicates a peripheral vestibular lesion. Rotational chair testing may also be used although the usefulness of this test is still hotly debated as it cannot give site of lesion. It only indicates that there is indeed a discrepancy in the vestibular system.

A very popular test of late is the computerized moving platform posturography (MPP) device. This device simply controls somatic sensory and visual cues allowing for the evaluation of the vestibular spinal system in isolation. There are six conditions associated with this standard MPP. The first three involve standing on the rigid platform. Visual input is then manipulated so that the eyes are opened for Condition 1, closed for Condition 2 (much like a Romberg test) and then opened but unable to fixate sense of visual surround moves and coordination with body sway. Con-

dition 3. The next three maneuvers repeat the visual manipulation but this time the platform is sway referenced to eliminate the somatic sensory input from the feet. Computerized posturography may be impractical in many office settings due to the technical and financial constraints. We feel these conditions may be easily assessed in the office with a thick piece of foam and a Chinese lantern that may be placed over the patient's head. This testing is normally referred to as "Foam and Dome" testing. The Romberg test mimics Conditions 1 and 2. By placing the lantern over the patient's head, one can mimic Condition 3 of MPP because the patient receives no fixed visual support and cannot fixate gaze. The patient is then asked to repeat Conditions 1-3 by standing on the thick foam for 20-30 seconds for each condition. The foam essentially eliminates somatic sensory input and we are able to test the vestibular system in isolation much in the same manner as MPP. In a recent prospective study by the third author; the sensitivity and specificity of foam posturography versus MPP demonstrated that the most common abnormalities were those for Conditions 5 and 6. This is to be expected since these two conditions rely solely on the vestibular system for information pertaining to balance. The sensitivity and specificity of the Foam and Dome test is 95 percent and the 80 percent

respectively for patients with peripheral vestibular lesions and had a confidence limit greater than 95 percent for the controlled patients. Thus Foam and Dome testing is felt to be a practical alternative in the office for those who do not have access to MPP.

Radiologic testing at times is essential in evaluating patients with dizziness and vertigo complaints. The test of choice is a MRI scan with gadolinium which will almost always be able to demonstrate any type of lesion along the vestibular auditory neural complex. Where this may deviate is those patients who have a history of trauma. In those cases computerized tomography or CT scan with fine resolution cuts of the temporal bones is useful in evaluating the integrity of the temporal bone.

#### **SUMMARY**

In conclusion, the assessment of the dizzy patient is indeed complex and far-reaching. The expertise of family practitioners, internist, neurologists, otolaryngologists, and neurosurgeons at times is essential and one should not feel inhibited about referring the patient for further evaluation to our colleagues so that the patient may best be served and resolution of the patient's complaints may result. □

# Editorials

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It is a pleasure to co-edit this issue of *The Journal of the South Carolina Medical Association* with its focus on Otolaryngology-Head and Neck Surgery. Our specialty, like all others in medicine, is undergoing relatively furious changes in patient referral patterns, physician autonomy and reimbursement for services. These changes are the source of debates, finger pointing and a great deal of anxiety for all of us. I deal with residents in training every day and hear them ask just as many questions about practice plans, billing, and CPT coding as on the reasons for ordering a given test or performing a given procedure. This is the reality that they perceive for themselves when they finish their training. I am in no way minimizing their concerns, particularly in light of what they hear and see in the press and in the hospitals and offices. Through this issue, and with the help of my co-editor and co-authors, I would like to offer a perspective on what really counts – the patients we treat. The financial concerns, endless paperwork and faltering image that physicians now have are superseded for me daily by the gratification of helping our

patients. This may sound trite and naive but the reality is that all the business problems will be there after this current wave of “managed care” passes and the next one gains momentum. Our patients with head and neck cancer, the children with congenital deformities and those with all the other ailments we treat will also still be there and will expect to be taken care of. One can argue (and we do) about responsibility, liability and who-is-going-to-pay-for-what but the clear decision is that the vast majority of us will continue to take care of patients regardless.

Otolaryngology-head and neck surgery is as comprehensive a field as exists in medicine. There is an opportunity to practice primary, secondary and tertiary pediatric, adult and geriatric care. I hope this issue gives the readers a glimpse into our specialty and the information presented is useful to you and your patients.

Marcelo Hochman, M. D.

Director, Facial Plastic and Reconstructive Surgery, Department of Otolaryngology  
Medical University of South Carolina  
171 Ashley Avenue, Charleston, SC 29425



# On the Cover:

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The cover illustration is from a hand-colored lithograph by Dr. J. M. Bougery published in Paris in 1832 (*Traite Complet de L'Anatomie de L'Homme*). Lithography, "to draw on stone," was introduced by Sennefelder in Germany in 1796 as a less costly alternative to the copper plate engraving. The image was drawn on finely polished limestone with a greasy ink and then a thin layer of water was poured onto the tablet. Paper was then pressed against the stone, and the elevated ink image was transferred and the non-image area was wetted with water. When the paper dried, the lithograph could be colored by hand. The image of this lithograph was probably inked on zinc, which was an improvement from the fragile limestone. Mechanization of lithograph coloring was introduced in the 1830s and utilized successive pressings with separate palates for each color. The original Currier and Ives pastoral scenes were printed with such a process.

The early anatomists and surgeons were one and the same. As structural and functional relationships were defined, surgical techniques were created or altered accordingly.

The first oncologically-sound operation for cervical metastases, the en bloc neck dissection introduced by Crile in 1906 and subsequently refined by Hayes Martin in the 1940s, was based on careful study of the cervical lymphatics. As revealed by the cover lithograph, these lymphatics were not well delineated in the mid-19th century. The newer "conservative" neck dissections which remove specific cervical lymphatics while preserving the internal jugular vein, spinal accessory nerve and/or sternocleidomastoid muscle were also developed in the anatomy and autopsy laboratories prior to use on cancer patients. Future developments in surgery will continue to depend on cooperation between the anatomist, pathologist, and surgeon.

M. Hochman, M. D.

J. David Osguthorpe, M. D.

Guest Editors

Department of Otolaryngology &  
Communicative Sciences

MUSC, Charleston, SC 29425



# Alliance Page

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## LEGISLATIVE AFFAIRS 1994-95

It is clear that health system reform issues will be back on the legislative agenda, both on the national and state levels. Many policy advisors, analysts and other healthcare consultants predict that the real reform will occur on the state level. The best and worst reforms could indeed happen at the hands of our new governor and state legislators. To this end, the alliance is working with your state medical association to develop a "Grassroots Network" of alliance members who are willing to serve as "on-call" advocates for the lobbying staff of the SCMA. The details of this program are still under development. What is clear is that the alliance is a group of your strongest allies, over 2,000 strong who could and should speak out for the best interests of you and your patients.

Among other legislative efforts this year is the voter registration drive. Jury duty lists are generated via the driver's registration data bank. **VOTER REGISTRATION IS NO LONGER THE MECHANISM FOR SELECTING CITIZENS FOR JURY DUTY.** So please register to vote, if you have not done so already. If you don't speak up for yourself and your spouse doesn't have time either, then who will be looking out for you? Stay informed this year and let your opinion be known. The South Carolina Medical Association Alliance is dedicated to advocating for you on the legislative front this year more than ever before. Help us help you! Thank you.

Margarita M. Pate, Chairman, Legislative Affairs  
Stephanie Evans, Co-Chairman, Legislative Affairs

---

# AMA POWER Network offers toll-free hotline on reform

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This service is part of the POWER (Physicians Organized to Work for Effective Reform) Network. Each day, the AMA will update the POWER Network hotline with the latest information on the House and Senate floor debate. Callers will hear a message describing floor activity, its impact on organized medicine and clear instructions on how they can have maximum impact on important upcoming votes.

Physicians and their families are encouraged to contact their senators and representatives to seek support for organized medicine.

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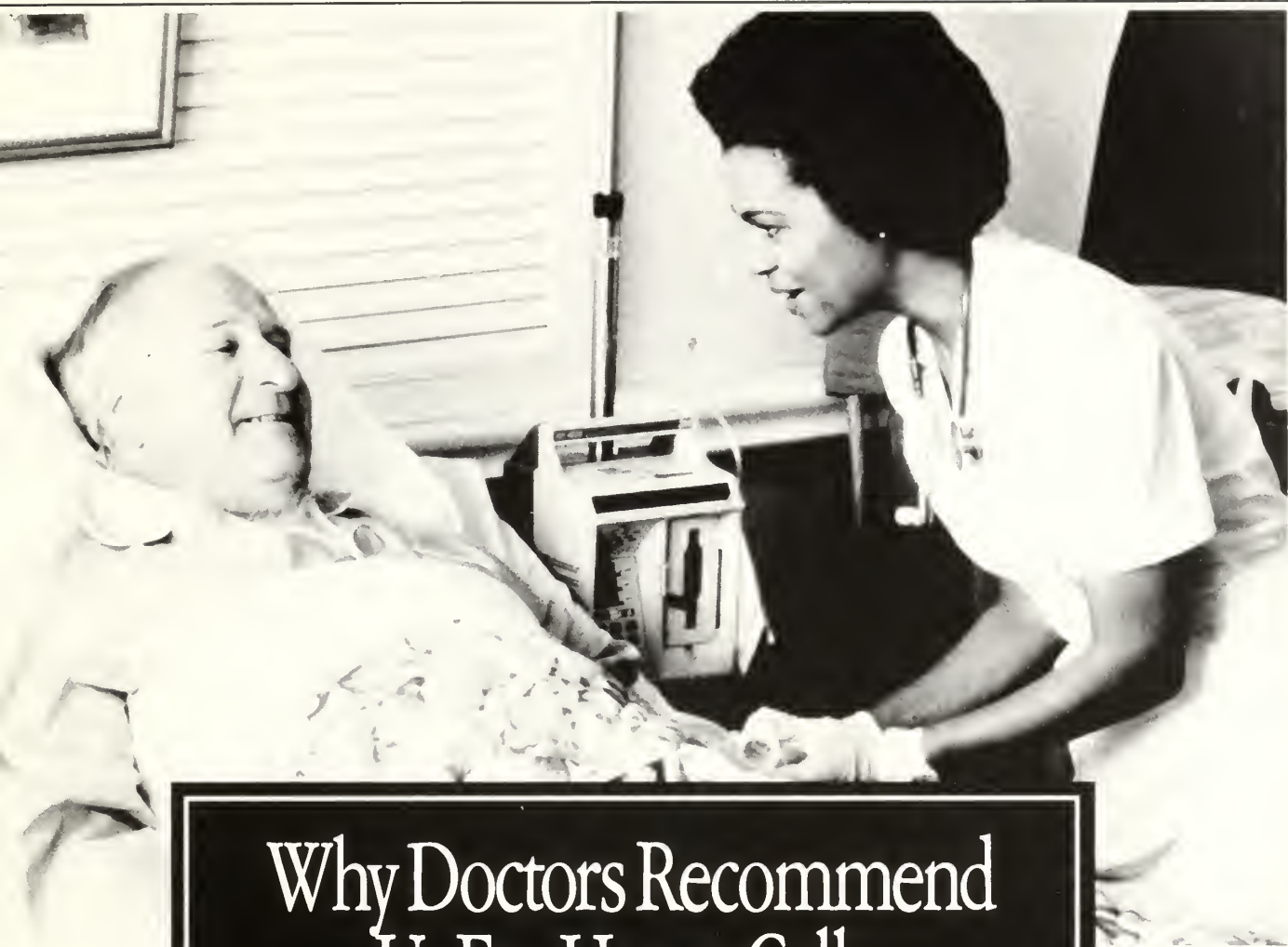
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## INDEX TO ADVERTISERS

B & B X-Ray .....	498
Baptist Medical Center .....	474
Anne M. Bowen, C. F. P. ....	Cover 4
The Cliffs at Glassy .....	516
Companion Technologies .....	443
Dewees Island.....	504
Harry A. Hancock, LL. M. ....	Cover 4
Interim Health Care .....	Cover 3
Lexus of Charleston.....	497
The Medical Protective Company .....	490
Nationsbank .....	444
Ultimate Auto Connection.....	494
U. S. Air Force Reserve.....	494
U. S. Army Active .....	482
U. S. Army Reserve.....	503
Winchester Surgical Supply Company .....	454
York X-Ray.....	Cover 2



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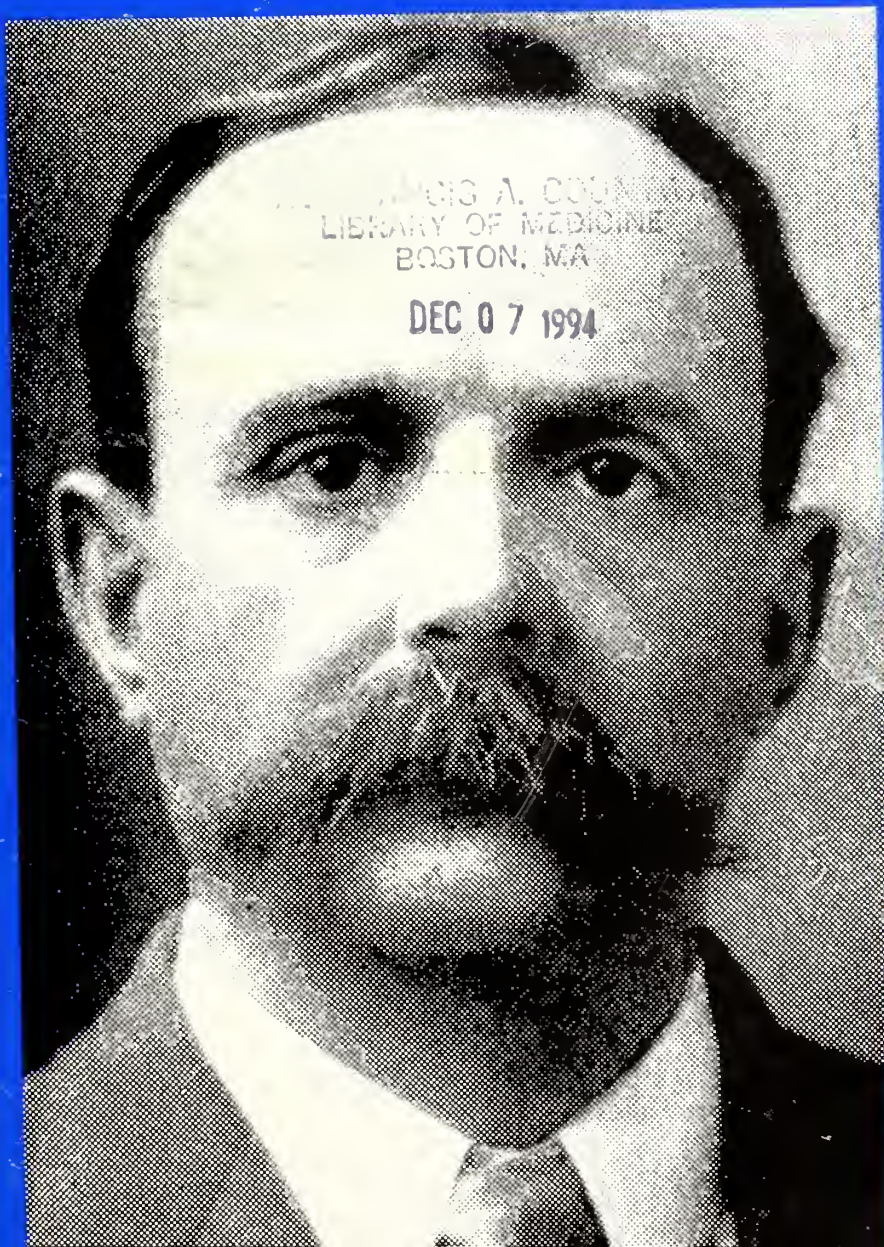
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PART 2**

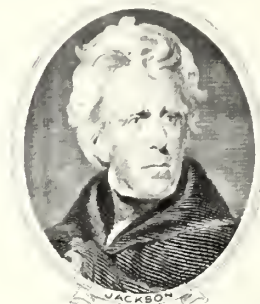
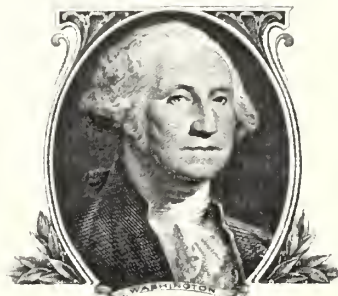
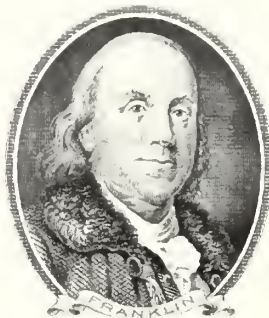
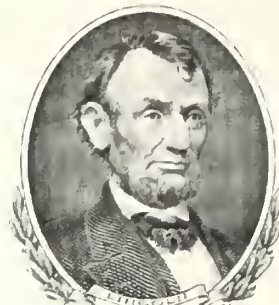
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# President's Page

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*O. Marion Burton MD*

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## Contents

*Special Issue: Alzheimer's Disease: Part 2*

*Guest Editor: Charles N. Still, M. D.*

### 533 Introduction

*Charles N. Still, M. D.*

### 534 Alzheimer's Disease and the Eye

*Mitchell Jay Wolin, M. D.*

### 539 Multi-Infarct Dementia (MID)

*Michael J. Malone, M. D.*

### 543 Treatment Strategies for the Cognitive Deficits of Alzheimer's Disease

*Kimford J. Meador, M. D.*

### 553 New Approaches in the Treatment of Alzheimer's Disease

*Jacobo E. Mintzer, M. D., David Bachman, M. D.*

### 560 Aluminum Neurotoxicity and Alzheimer's Disease

*Charles N. Still, M. D.*

## Features

### 571 Alliance Page

### 569 On the Cover

### 531 President's Page

## Association

### 565 Gray Matter

### 549 SCMA Newsletter

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## SPECIAL ISSUE: ALZHEIMER'S DISEASE

GUEST EDITOR: CHARLES N. STILL, M. D.\*

### PART 2: INTRODUCTION

---

In this issue of *The Journal of the South Carolina Medical Association*, we present the second group of authors on Alzheimer's disease (AD). Dr. Mitchell Wolin provides readers a lucid account of visual disorders which may occur in AD. Dr. Michael Malone gives a concise review of multi-infarct dementia and its relationships with AD. Dr. Kimford Meador offers new insights into the complex issues involved in developing successful treatment strategies for the cognitive deficits of AD, including the use of high-dose thiamine. Dr. Jacobo Mintzer and Dr. David Bachman give us new approaches to the comprehensive management of the cognitive and behavioral disturbances of AD, including the importance of caregiver support. In the final paper, I address the issue of aluminum neurotoxicity and its possible association with AD.

We hope that you will find Part 2 just as useful and interesting as Part 1. As Guest Editor I wish to thank each of the contributing authors, as follows:

Ruth K. Abramson, Ph. D.  
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Mitchell Jay Wolin, M. D.  
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I also wish to thank the Editor of *The Journal*, Dr. Charles S. Bryan; the Managing Editor, Ms. Joy Drennen; and my colleagues of the Editorial Board for their support of these special issues to honor the memory of Professor Dr. Alois Alzheimer on the 130th anniversary of his birth. Prosit! ☐

---

\*W. Jennings Bryan Dorn VA Hospital, Columbia, SC 29201.

# ALZHEIMER'S DISEASE AND THE EYE\*

MITCHELL JAY WOLIN, M. D.\*\*

"I'm having trouble reading." This is an extremely common complaint, but what do we do when the patient's exam is normal? First of all, we must understand what is meant by *normal*. The typical ophthalmologic examination does not specifically test integrative visual functions, which require higher cortical interpretation; this is frequently where such patients show difficulty. Awareness of the various ocular problems that Alzheimer's disease (AD) patients may have, even in the presence of a normal standard eye exam, is extremely important in reaching the proper diagnosis, and possibly in enabling treatment. A few simple tests can be incorporated into the exam, if necessary. Utilizing a few tests that evaluate visual integrative function will help the clinician understand the complaints of visually symptomatic Alzheimer's patients.

## VISUAL ASSOCIATION AREA DAMAGE

Most acuity tests require the patient to read individual letters. There is no routine test done to assess a person's ability to make understandable words out of the letters. Therefore, it can be very helpful to have the patient with possible AD read a paragraph aloud. A patient may have nearly normal acuity, and yet have great difficulty in reading a paragraph. Trobe describes a 69-year-old retired engineer who complained that he could not read. He noted that objects would "pop in and out of my sight." He often had *more* difficulty with large headlines than with ordinary newsprint. Upon reading a paragraph, the patient stumbled in several ways.

He would wander off onto the wrong line; stop unpredictably, as if blocked; and misread simple words. Although his acuity and fields were mildly abnormal, his difficulty in reading far exceeded what one would expect.<sup>1</sup>

Studies have demonstrated defects in AD patients in the following areas: Visuospatial memory, visuoconstructional ability, visuo-perceptual ability, visuospatial learning, facial recognition and facial recognition memory.<sup>2</sup> Let us try to define these vague terms. *Visuospatial memory* refers to a patient's ability to retain a mental image of his surroundings, and to successfully navigate through the environment. An elderly woman may have difficulty in finding her way back to her bedroom from the bathroom while at her son's house. *Visuoconstructional ability* is tested by whether or not a patient can copy simple geometrical figures. *Visuoperceptual ability* generally is used to describe an individual's sophistication in describing a complex picture. The patient may be able to describe individual elements in a picture, but not be able to integrate them into a whole meaning. For example, a patient may describe different parts of a picture, such as a horse, man, and field, but not be able to realize that it is a picture of a farm. From a practical standpoint, one may show a patient a complex picture from a magazine and ask the patient to describe it. When the response is abnormal, this is commonly called *simultanagnosia*. This may be isolated, or be a part of Balint's syndrome, which may be seen in AD. These patients have difficulty in exploring space, poor hand-eye coordination (optic ataxia), and abnormal control of eye movements (psychic paralysis of gaze).<sup>3</sup>

How do we obtain visual information about a picture or scene? We utilize what has been referred to as a *visual scanpath*. It has been

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demonstrated that there are specific visual strategies that subjects use to examine an informative visual scene. This is the combination of vertical and horizontal eye movements used to look at the various parts of the scene. AD patients may have *visuospatial dysgnosia*, and therefore may have abnormal scanpaths. With such a problem, there will be no substantial strategy of gaze.<sup>2</sup> A patient's level of confusion in trying to interpret a picture will be increased, possibly because of incomplete visual information. This may contribute to *simultanagnosia*. There also appears to be a slowing of the time needed to encode visual information.

*Visuospatial learning* has been tested in patients by comparing their abilities utilizing visual versus auditory clues. Facial recognition refers to the ability of patients to recognize familiar faces, a complex task. A deficit of facial recognition is referred to as *prosopagnosia*. Facial recognition memory can be tested by showing patients a picture of a face and having them identify the similar face in a group of photographs subsequently presented.

Trobe et al have recently devised a series of clinical tests to allow easier and more accurate diagnosis of the visual problems of AD patients.<sup>4</sup> These tests are as follows:

1. Identification of line drawings, such as eyeglasses, a cat, an airplane;
2. Identification of three line drawings that overlap each other;
3. Identification of line drawings "masked" by hatched lines drawn over the picture
4. Identification of a line drawing that is broken up into three parts on the page (fragmented forms). (I might add that I had some trouble with this one! It was a hoe, something that I never saw in my New York suburban childhood!);
5. Having the patient place cross marks on each of multiple short lines on a test page (line cancellation);
6. Line cancellation with a higher density of lines on the page;
7. Reading a paragraph;

#### 8. Putting together a simple puzzle.<sup>4</sup>

While this battery of tests may eventually be commercially available, at present, one can create an appropriate group of tests when needed, keeping in mind the ideas discussed above.

Pathologically, neuronal degeneration, neurofibrillary tangles, and neuritic plaques have been discovered in the visual association cortex and high-order visual association cortex.<sup>5</sup> Biochemical assay of AD associated protein was described by Ghanbari et al. They utilized ALZ-50, a monoclonal antibody that reacts against brain tissue homogenated from AD patients. They state that this biochemical assay is a useful adjunct to the clinicopathological diagnosis of AD.<sup>6</sup>

### CONTRAST SENSITIVITY

The ophthalmologist typically tests visual acuity with a distance Snellen chart, which has dark letters against a white background. This is a high contrast situation; patients with mild defects of contrast sensitivity will be missed (a "false negative"). Similarly, near vision is tested with a reading card at 14 inches, using the patient's bifocals. This is also a high contrast test. Contrast sensitivity may need to be specifically tested by utilizing gratings of light and dark bars, and by measuring the patient's ability to distinguish the gratings.

Overall loss of contrast sensitivity is consistent with frequent complaints of poor vision in AD patients. Whether this loss occurs more at high frequency or at low frequency is still debatable.<sup>6,7</sup>

### COLOR VISION

*Dyschromatopsia*, or abnormal color vision, may be one manifestation of the optic neuropathy of AD. It has been noted that AD patients often are unable to perform well with color test plates, which possibly can be attributed to visuospatial difficulties instead of simple color deficiencies.<sup>1</sup> However, on Farnsworth-Munsell testing, it has been shown that AD patients may be able to name

the colors, but are unable to arrange them in proper order.<sup>8</sup>

## STEREO VISION

Cronin-Golomb evaluated stereoacuity in AD patients utilizing the Randot Stereo Acuity test. They attributed the poor performance of their patients to the known pathological status of extrastriate cortex in this disorder. It was noted that previous studies have suggested that the right hemisphere has a greater impact on stereoacuity than the left hemisphere.<sup>9</sup>

## ABNORMALITIES OF SACCADIES

There are numerous other abnormalities of ocular function that can occur due to AD. These can give visual symptoms, but the patient may not be easily able to describe them. Fixation instability is a common finding. A normal individual will exhibit a small number of what are referred to as *saccadic intrusions* (square wave jerks or SWJs). Typical saccades are voluntary fast eye movements, such as when looking from one object to another. SWJs are small saccades which are involuntary breaks off fixation that occur up to six times per minute in normals. (specifically, their amplitude is 1.75 to 6.5 degrees). An increased frequency of saccadic intrusions in AD patients can be correlated with the severity of the dementia.<sup>10</sup> In addition, forcing a patient to fixate often will increase the frequency of the saccadic intrusions. Patients with symptomatic square wave jerks may possibly benefit by treatment with methylphenidate (Ritalin).<sup>11</sup>

*Saccadic latency* is a difficulty with the amount of time it takes to initiate fixation on a desired target, and is controlled by a complex process in the brain. This difficulty with triggering of saccades is correlated with overall disease severity, but not directly with dementia alone.<sup>2</sup> The delay in saccadic latency can be accurately measured with eye movement recording equipment.

## PURSUIT EYE MOVEMENTS

Pursuit (or slow) eye movements are often

abnormal as well. Pursuit eye movements are used to follow objects in motion. With loss of normal smooth pursuit, catch-up saccades are required. In AD patients, the frequency of catch-up saccades is increased, giving a choppy appearance to the pursuit. The ratio of eye velocity to target velocity, known as mean pursuit gain, is abnormally low, so that catch up saccades must be utilized to keep the eye following the target.<sup>12</sup>

## ANTISACCADES

An interesting research test is the anti-saccade. The patient is instructed not to look at a flashed target, but rather to look at an equal distance in the opposite direction. AD patients often have great difficulty with this, presumably due to a loss of ability to suppress the superior colliculus. This has been referred to as the *visual grasp reflex* in AD.<sup>2</sup>

## OPTIC ATROPHY

There is some controversy in the literature as to whether or not optic atrophy and concomitant ganglion cell loss occur as part of AD. According to Tsai et al, AD patients had detectable nerve fiber layer damage in the retina (implying damage to retinal ganglion cells), utilizing red free photography. There was also an increase in cupping of the optic disc in their patients.<sup>5</sup> These findings have been variable in studies by other authors, and therefore there is still controversy as to whether or not clinically significant optic atrophy can be solely attributed to AD.

It has been noted by Triek et al that AD patients may show specific defects in motion, assessed by dynamic random dot displays. These functions are particularly dependent on what is referred to as the M-cell system of the retinal ganglion cells.<sup>13</sup>

## ELECTRORETINOGRAPHY (ERG) AND VISUAL EVOKED POTENTIAL (VEP) ABNORMALITIES

There are conflicting results in studies of ERGs in AD. The pattern electroretinogram (PERG) can show low amplitude compared to

normals. The flash ERG is normal. It is felt that this difference is due to abnormalities of the retinal ganglion cells and axonal depletion of the optic nerve.<sup>2</sup> Again, these results were not consistently found by other authors.

The flash VEP P2 wave is thought to reflect the integrity of higher cortical areas, whereas the pattern VEP is felt to be generated in the primary visual cortex. A delayed VEP P2 has been shown to occur in AD, and its latency increases throughout the course of the disease.<sup>2</sup>

### SPECT AND PET SCANS OF VISUAL PARTS OF THE BRAIN

Positron emission tomography (PET) studies have shown that cerebral hemispheric hypometabolism is not evenly distributed in AD patients. Rather, it is most evident in the posterior parietal lobes and adjoining temporal and occipital lobes. Patients with significant visuospatial dysfunction typically will have relative hypometabolism in the right parietal lobe.<sup>1</sup>

In Balint's syndrome, single photon emission computed tomography (SPECT) and positron emission tomography (PET) may show hypoperfusion and hypometabolism in the posterior parietotemporal regions, with relative sparing of the visual cortex.<sup>3</sup>

### TREATMENT

Biochemical changes in the brains of AD patients include a reduction in the muscarinic and nicotinic receptors, and decreased activity of choline acetyltransferase and acetylcholinesterase. Tacrine hydrochloride is a new, centrally active, reversible cholinesterase inhibitor. It is the first drug approved by the FDA for the treatment of AD. It increases release of acetylcholine, and is also believed to have multiple effects on several other neurotransmitter systems. Significant functional improvements with the use of tacrine have been shown in some studies, but these evaluations are difficult to perform. Side effects of tacrine may be significant, including transaminase elevation, nausea and

vomiting, diarrhea, and skin rash.<sup>14</sup> Tacrine can be used in combination with lecithin. The typical dose of tacrine is 80 mg/day at an approximate cost of \$110 per month. Weekly transaminase levels should be monitored until the daily dosage has been stabilized. Most studies have looked at cognitive impairment, and exact measurements of visual functions in patients on this medication have not been reported.

### SUMMARY

AD patients may have substantial defects in the visual system despite a normal routine eye exam. Specific methods should be utilized to assess the ability of these patients to understand what they see and read. Subtle defects of eye movements may be present. Problems with contrast sensitivity, color vision, and stereo vision may also be present, as well as early signs of optic atrophy. An awareness of the specific problems that AD patients present will lead the clinician to accurate diagnosis.

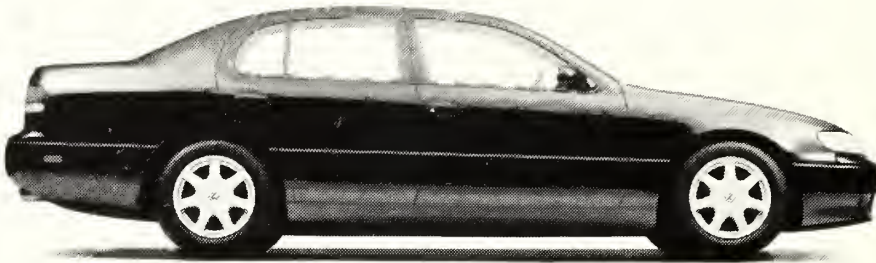
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# MULTI-INFARCT DEMENTIA (MID)

MICHAEL J. MALONE, M.D.\*

---

In the period 1985 to 2000 in South Carolina the population over age 65 years is projected to increase from 350,282 to 484,104. This is a 38 percent increment within a 15-year period. By the year 2000, 117,153 black individuals over age 65 will be living in South Carolina.<sup>1</sup> These individuals, both men and women, have a higher prevalence rate than whites for hypertensive vascular disease and this higher rate increases with age from the second to the seventh decade of life.<sup>2</sup> The increased prevalence of hypertensive vascular disease is also associated with an increased prevalence of cerebral vascular disease and stroke with age in this population.<sup>3</sup> Although Senile Dementia of the Alzheimer Type (SDAT) is the most common late-life dementia,<sup>4</sup> vascular dementia due to multiple small strokes (MID) is in second place.<sup>5</sup> The prevalence of MID in the age group over 65 years has been estimated from eight percent<sup>3</sup> to 12 percent.<sup>4</sup> The frequency of vascular forms of dementia has been reported as high as 34 percent in some clinical series.<sup>4</sup> In terms of all progressive dementias over age 65, SDAT accounts for 55 percent, MID 20 percent, SDAT + MID 15 percent and "all other" 10 percent of the total number of cases.<sup>8</sup> However, these percentages are based on epidemiological data from a large northern urban population. In South Carolina, the incidence of SDAT and MID may be reversed from Mortimer's figures.

The term "Multi-infarct Dementia" was coined by Hachinski, Lassen and Marshall in 1974<sup>9</sup> but the concept that multiple small strokes could produce mental status change was popularized much earlier by Alvarez.<sup>10</sup>

## VULNERABILITY

A key issue in MID is the total volume of brain tissue infarcted. The loss of 50-100 cc is a certain index of dementia.<sup>11</sup> The loss of five to 10 percent total brain mass in a diffuse fashion results in irreversible dementia. Dementia, the loss of cognitive function, occurs as a symptom of tissue loss. A diffuse tissue loss of this magnitude in a widespread, rather than local fashion through the brain takes place because cerebral tissues are vulnerable. This vulnerability arises because the energy demands of brain are massive and continuous. Central nervous system tissues require a continuous supply of glucose and oxygen through the cerebral blood flow. Unlike other tissues and organs, energy reserves in brain are minimal.

Glycogen is present in brain as a very limited energy reserve (3.3 mmol/mg), primarily in astrocytes, and constitutes less than two percent of the glycolytic flux.<sup>11</sup> In intact brain tissue, as contrasted with slices and homogenates, the only significant energy substrate is glucose. Glucose metabolism or glycolysis is regulated primarily by the enzymes hexokinase and phosphofructokinase.<sup>12,13</sup> Glycolysis generates high-energy (33 equivalents per mole glucose) phosphate (~P) bonds which are regulated in turn by the ADP-ATP (adenosine di- and tri-phosphate) flux. Another system, CPK (creatine phosphokinase) is even higher than ATP in brain and provides ~P for phosphorylation of ADP. The CPK system is also critical in regulation of mitochondrial function in neurons. About 5.8 percent of brain glucose is metabolized via the pentose shunt pathway.<sup>14</sup> This hexose monophosphate shunt is particularly active during development, producing the NADPH required for reductive reactions in lipid synthesis and myelin biosynthesis.

---

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Metabolically, the brain is one of the most active tissues in the body. In young adults, the brain requires 3.5 ml  $O_2$ /100 g/min (49 ml/1400 g/min). An average man, weighing 70 Kg requires 250 ml  $O_2$ /min. The brain, although only two percent of the body weight, requires 20 percent of the total resting  $O_2$  requirement.<sup>15</sup> Oxygen in the brain is used almost entirely for the oxidation of carbohydrate. This involves the turnover of 7 mmol or  $4 \times 10^{21}$  molecules of ATP/min. This enormous energy requirement is needed almost solely for excitation and conduction. Most of brain energy consumption supports ion transport and sustains membrane potentials. Both are unceasing in brain. Beyond early development, comparatively little energy is devoted to tissue synthesis and breakdown. Although there are some brain proteins with relatively short half lives and neurotransmitter synthesis/degradation is energy-requiring, these functions utilize only a minute fraction of brain-energy.<sup>16</sup> This large oxygen demand and glucose consumption (3.5 mg/100g/min) require a correspondingly large blood flow and this is estimated at 57 ml/100 g/min (798 ml/whole brain/min) or 15 percent of the total cardiac output in the resting state.

Regional control of blood flow at the arteriolar level is determined by local chemical changes ( $pCO_2$ ,  $pO_2$ , pH) and other unknown (functional) factors. Local cerebral metabolism may be measured by isotopic methods ( $^{14}C$  Deoxyglucose) involving blood A/V (arteriovenous) differences in the experimental animal or by  $^{18}F$  Fluorodeoxyglucose, (a gamma emitter) in PET [Positron emission tomography] scanning techniques. Normally, cerebral blood flow and oxygen consumption change very little in humans from early adulthood into normal old age. However, where blood flow and perfusion is reduced in arteriosclerosis, significant regional decrements in the cerebral metabolic rate (CMR) for oxygen and Glucose appear.

### CLINICAL PRESENTATION

Failure of cerebral blood flow in the face of

continuing energy demand, i.e. in the normal active or basal state, leads to cerebral infarction. The extent of tissue damage is dependent on duration of the deficit and the size of the vascular area. In classic stroke syndromes subsequent to obstruction in extracranial arterial supply, tissue damage will follow the perfusion areas of major cerebral vessels or the "border zones" between these vessels. These vascular injuries have recognizable clinical parameters: typically a sudden onset; symptoms restricted to known vascular territories, with paresis, sensory loss, aphasia, visual field deficits; in vertebrobasilar insufficiency, brain stem or posterior fossa signs – an alteration in consciousness, nausea, vomiting, unsteadiness, diplopia. After a latency period of 12 to 48 hours, most larger infarctions will be demonstrable as hypodense areas on CT scans. These large arterial strokes may be associated with dementia, particularly when they are bilateral and/or involve extensive tissue damage. In such cases, dementia is a symptom with other localizable findings.

Another category of patients – those with MID – show a very different clinical picture. The onset of symptoms is insidious rather than abrupt, and commonly involves behavioral changes. Attempts by family members to date the onset of symptoms are difficult and uncertain. They usually reference only significant social events such as holidays. These are poorly related to the appearance of earliest symptoms. Maladaptive behaviors with personality change and memory loss may bring the patient to medical attention. Emotional lability is common. In early stages, persistent insight into intellectual loss may precipitate a significant degree of depression. Typically, the patient loses interest in current events, and with memory deterioration may not recognize familiar places or persons. In many cases, the progressive dementia is associated with language difficulties and aphasia may inhibit communication. Visuo-spatial problems are common; the patient can become lost in previously familiar surroundings. These changes are not dissimilar from



the symptoms of Alzheimer's Disease, but the clinical course of MID patients tends to be a stepwise deterioration rather than a continuous down hill regression. Further, the patients often experience periods of improvement early in their course, particularly after an acute exacerbation. On neurological examination, mental status changes are evident, and these may be striking to observers who are familiar with the patient's premorbid state. Problems are evident when the patient is tested for use of abstract concepts or similarities/dissimilarities. The fund of general knowledge shrinks and the patient may be unaware of major current events. Memory functions are impaired, particularly recent memory, but remote memory is usually abnormal in terms of memory associations. Immediate memory (digit sequence) is usually intact. Until near terminal stages, the patient is usually aware of self and location, and that something is wrong. There are usually few cranial nerve signs but the patient's speech may be slurred and dysarthric. On motor system examination, gross paresis may be absent but there may be changes in tonus, and reflexes; Babinski signs may be present. A presentation with plastic hypertonus and bradykinesia may resemble Parkinsonism in some individuals.

A major risk factor for MID is hypertension, particularly when long standing and poorly controlled. Diabetes mellitus also may be an etiological factor. MID cases do not differ in any important respects from the diffuse cortical involvement described earlier by Binswanger<sup>18</sup> as a complication of hypertensive encephalopathy.

## **PATHOLOGY**

The major neuropathology of MID consists of multiple small lacunes (2-15 mm in diameter). The lacunes form after a degenerative process (lipohyalinosis) destroys the walls of small arterioles, usually as a consequence of long standing hypertension. Subintimal deposits of a hyalin fibroid substance narrow the vessels walls and lead to thromboses of

their lumina<sup>19</sup> with minute infarction of the surrounding tissue. Once reabsorbed, these minute infarctions result in the lacunar cavities. Mural destruction and formation of microaneurysms also occurs. These can rupture and result in small hemorrhagic extravasations from the blood vessels with perivascular accumulation of hemosiderin-filled macrophages. Rupture of larger aneurysms can result in intracerebral hemorrhages.<sup>20</sup> Roman<sup>19</sup> has noted that the number and size of lacunes does not correlate well with the degree of dementia.

MID represents a progressive process, usually insidious in onset, characterized pathologically by multiple small vessel obstructions with microinfarcts. Neurological examination reveals mental status changes and often transient, "soft" neurological signs. Higher cortical function deficits (apraxias, aphasias, agnosias) may be present but the CT scan results are usually non-specific (atrophy, ventricular enlargement) or normal. In contrast to CT, NMR (Nuclear Magnetic Resonance – MRI) imaging may be far more sensitive and specific for the early changes of MID.<sup>21,22</sup>

## **MANAGEMENT**

Prevention of further decline and stabilization of functional level are important goals in treatment of patients with MID. Early suspicion, careful evaluation and attention to known risk factors – hypertension and diabetes – are critical. In this context, attention must be given to mean arterial pressure (MAP) in patients with grossly elevated systolic and relatively normal diastolic levels. However, overly aggressive anti-hypertensive therapy with attendant orthostatic episodes may exacerbate cerebral perfusion deficits in border zone areas. The possibility that serum lipid alterations may play a role in the pathogenesis of MID,<sup>23</sup> suggests that dietary measures and moderate exercise are useful, analogous to the treatment of coronary arterial disease. Patients with MID are elderly and very sensitive to neuroleptic medications. Benzo-

diazepines particularly should be used cautiously and in low dosage; sedatives should be short-acting and used only when absolutely necessary. These patients will do best in a familiar setting; home care, whenever possible, is highly desirable. □

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# TREATMENT STRATEGIES FOR THE COGNITIVE DEFICITS OF ALZHEIMER'S DISEASE

KIMFORD J. MEADOR, M. D.\*

Alzheimer's disease is a devastating disease for both patients and their families who are often desperate for help and want their loved one to receive the latest treatment. Thus, a physician may be confronted by the patient's family with news reports of "miracle cures." The physician is left with the task of explaining the scientific results of these studies, placing the results in a proper clinical perspective, and advising the family as to availability and possible alternatives.

## ASSESSING CLINICAL RESEARCH RESULTS

Many clinical trials in Alzheimer's disease are difficult to interpret.<sup>1</sup> The design and conduct of such studies are fraught with problems. First, there is no biologic marker for Alzheimer's disease, and a definite diagnosis requires brain tissue. Therefore, patients are enrolled with a clinical diagnosis of "probable Alzheimer's disease" or "dementia of Alzheimer's type" which carries a 15 percent-age misdiagnosis rate. Further, Alzheimer's disease appears to be a disease syndrome with multiple contributing etiologies and with different subtypes manifesting varied clinical presentations. Targeting treatment to a single sign or symptom is problematic, and psychometric evaluation of patients in this population is difficult. Differences in study design may confound comparisons between studies (e.g., carry-over effects in crossover studies, treatment duration, outcome measures, and subject selection bias). Reports indicating improvement on a drug in one study frequently are not confirmed by subsequent investigations. These replication failures may be due to differences in experimental design, a true

lack of efficacy, or an inadequate sample size to demonstrate a mild beneficial effect. Finally, the results of clinical trials need to be assessed not only for statistical significance but also for clinical significance. To date, only mild beneficial effects have been produced in any clinical trial.

## OVERVIEW OF THERAPEUTIC STRATEGIES

At the present time, Alzheimer's disease remains incurable. However, the disease is treatable in the sense that some modest therapeutic agents are available for the cognitive deficits, and psychotropic agents may be effective in controlling associated psychiatric problems. Further, the physician plays an important role in providing guidance in non-pharmacological management of the patient and in counseling the family on psychosocial and legal issues. Nevertheless, a satisfactory treatment of Alzheimer's disease is lacking, in large part due to our poor understanding of the basic disease mechanisms underlying the pathological changes. Continued basic research, as well as well controlled clinical trials, are critical to the development of improved therapies. Patient families have difficulty accepting double-blind, placebo-controlled investigations, especially for the long placebo treatment periods which are frequently required. In addition, many patients are excluded from controlled clinical studies because they have other diseases or risk factors for other types of dementia (e.g., vascular).

Treatment strategies for the cognitive deficits of Alzheimer's disease include symptomatic therapies to enhance cognition, treatments to slow or to arrest disease progression, therapies to delay disease onset, and interventions aimed at disease prevention. The cogni-

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tive deficits in Alzheimer's disease have been more closely linked to the loss of synaptic connections than to neurotransmitter abnormalities or other pathological lesions (e.g., plaques and tangles). Therefore, the development of a cure for Alzheimer's disease will ultimately require an intervention which halts or prevents cell death and the loss of synaptic networks. However, unless a completely effective prevention is devised, there will continue to be a role for symptomatic therapies.

### CHOLINERGIC THERAPIES

Observations of the importance of acetylcholine in memory mechanisms and the marked loss of cholinergic activity in the brains of patients with Alzheimer's disease have led to numerous attempts to enhance cognitive function in these patients by cholinergic manipulations. The acetylcholine precursors choline and lecithin (phosphatidylcholine) have not been effective. Acetyl-L-carnitine is a weak cholinergic agonist which may also serve as an acetylcholine precursor in the formation of acetyl CoA. It has shown some very mild beneficial symptomatic effects. Direct cholinergic agonists (e.g., arecoline) produce slight beneficial effects but in the past have required parental or intraventricular administration. A new cholinergic agonist (CI-979) which can produce CNS effects following oral administration is under investigation. Nicotinic agonists have not proven effective thus far, but new agonists (e.g., ABT-418) are in development.

In human trials, the most consistent, although modest, beneficial effects have been seen with cholinesterase inhibitors (e.g., physostigmine, tacrine, velacrine) which prolong the effects of synaptically released acetylcholine by inhibiting its breakdown.<sup>2,3</sup> Recently, tacrine (Cognex®) became the first drug approved by the FDA for the treatment of the cognitive deficits of dementia of Alzheimer's type. Tacrine produces a mild symptomatic improvement in the cognitive dysfunction of a subset (i.e., approximately 40 percent) of patients with dementia of

Alzheimer's type.<sup>4</sup> In those who respond, the improvement is roughly equivalent to reversing the deterioration of six to 12 months. The therapeutic effect is symptomatic, and patients continue to deteriorate. There has been a debate concerning whether this statistically significant improvement is clinically significant in view of the approximately 15 percent incidence of nausea (primarily at higher dosages) and 30 percent incidence of reversible hepatotoxicity which necessitates weekly monitoring of liver function (i.e., ALT) for about the first six months of therapy. However, tacrine is the only currently approved effective agent for dementia of Alzheimer's type. Further, a smaller subset of patients have a more robust improvement, and greater than 80 percent who developed the hepatotoxicity can eventually tolerate the drug because the hepatotoxicity usually does not recur on drug rechallenge.

### OTHER NEUROTRANSMITTERS

Alzheimer's disease also affects other neurotransmitters (i.e., norepinephrine, dopamine, serotonin, peptides) to a variable extent. Thus, treatment strategies which target other transmitters or manipulate multiple transmitter systems may be a rational approach to symptomatic therapy. Clonidine, an  $\alpha_2$  adrenergic agonist, produces mild beneficial effects in alcoholic dementia but no effects in patients with dementia of Alzheimer's type. However, the combination of clonidine and the anticholinesterase physostigmine is synergistic in enhancing cognitive performance in aged primates. Similarly, a new agent (busipirdine) with both adrenergic and cholinergic receptor activity is under investigation.

Methylphenidate and amphetamines, psychostimulants which increase central catecholamine turnover, are used by some physicians to treat dementia of Alzheimer's type, but have shown no benefit in well-designed studies. The dopaminergic agents levodopa and memantine have no benefit in dementia of Alzheimer's type. A synthetic analog of GABA (THIP), several peptide agonists (e.g.,

analogs of vasopressin and ACTH), and the opiate antagonist naloxone have been unsuccessful. Serotonergic antagonists have been noted to produce a mild enhancement of memory in some animal studies. Such an agent (ondansetron) is now being studied in patients with dementia of Alzheimer's type.

### **METABOLIC ENHANCERS**

Nootropics, such as piracetam and aniracetam, are a class of stimulant, psychoactive drugs which enhance cerebral metabolism. Their primary mode of action is uncertain but may be stimulation of the GABA system. When combined with cholinergics, nootropics appear to produce mild improvement in social behavior and attention. Dihydroergotoxine (Hydergine®), a cerebrovasodilator which theoretically enhances neuronal metabolism, has been used in the past to treat dementia. It is a safe drug which may lessen depressive symptoms but does not significantly improve cognitive function.<sup>4</sup> Similarly, other vasodilators and metabolic enhancers have not shown significant effects.

### **THIAMINE**

Thiamine-dependent enzymes are decreased in the brains of Alzheimer patients, and these enzymatic deficits exceed expected reductions secondary to neuronal loss and occur in regions relatively unaffected on histological examination. They are comparable in magnitude to the reductions in enzymatic markers of cholinergic activity. In addition to its important metabolic role, thiamine has a unique neurophysiological role in cholinergic systems, and appears to act as a modulator of cholinergic activity. In preliminary studies, high doses (e.g., 5 gm PO/day) of thiamine hydrochloride appear to produce a mild improvement in the cognitive function of patients with dementia of Alzheimer's type.<sup>5</sup> This effect appears to be symptomatic, with ultimate progression of the disease. It may be mediated cholinergically since high dose thiamine can partially reverse the adverse cognitive and EEG effects of the anticholinergic

agent scopolamine.<sup>6</sup>

### **NEUROPROTECTORS**

Since cognitive dysfunction in Alzheimer's disease is due primarily to loss of neurons and synapses, drugs which manipulate neurotransmitters or superficially enhance cerebral metabolism are unlikely to produce dramatic cognitive effects and will not halt the progressive deterioration. Treatment strategies directed at slowing the neuronal deterioration have been tried. L-deprenyl (selegiline), which reduces oxidation via monoamine oxidase B inhibition, has minimal effects on behavior in dementia of Alzheimer's type. These effects may be attributed to its antidepressant activity. L-deprenyl is also being studied in combination therapy with alpha-tocopherol, an antioxidant, in an attempt to reduce the progression of dementia of Alzheimer's type, but an early report is negative. Acetyl-L-carnitine has several actions including antiperoxidative effects, but has not been shown to slow progression. In one study, intramuscular desferrioxamine appeared to slow the progression of dementia in patients with probable Alzheimer's disease.<sup>7</sup> Desferrioxamine is a chelating agent which has been used for the treatment of aluminum and iron toxicity.

Neurotrophic factors are naturally occurring peptides that promote survival and enhance neuronal function. Although a deficit in neurotrophic factors has not been demonstrated in Alzheimer's disease, such factors might increase survival of diseased neurons or restore the activity of quiescent neurons. In Alzheimer's disease, there is a marked loss of neurons in the nucleus basalis of Meynert which provides the major cholinergic innervation to the neocortex. In animal studies, nerve growth factor has been shown to retard cell loss after lesions in this region.<sup>8</sup> Other neurotrophics may have similar restorative or protective properties. However, at present these factors require direct introduction in the central nervous system (e.g., via intraventricular pumps). Further, the long term effects are unknown.

Animal studies suggest that estrogen may be protective to cholinergic neurons, and these effects may be mediated by nerve growth factor. One study found that women on estrogen replacement therapy were 40 percent less likely to develop Alzheimer's disease, but another study found no consistent evidence of an effect of estrogen on cognitive function.

## AMYLOID

Amyloid is deposited throughout the brains of patients with Alzheimer's disease. One theory posits that a cascade of events in the proteolytic processing of the amyloid precursor protein leads to the abnormal deposition of amyloid which due to its toxicity results in the death of nerve cells.<sup>9</sup> Both genetic and environmental factors appear to influence the chances of plaque formation. Therapeutic strategies might include inhibiting amyloidogenesis or blocking amyloid neurotoxicity. The molecular genetics of amyloid formation is being intensively investigated and may lead to effective therapies. Substance P appears to block amyloid neurotoxicity, but effective means of drug delivery are not available. Uncontrolled stimulation of excitatory amino acids and calcium dysregulation can result in neurotoxic effects. Therefore, agents such as NMDA antagonists or calcium channel blockers may be of benefit.

Although Alzheimer's disease is not an inflammatory disease in the traditional sense, amyloid plaques are associated with local inflammatory responses. In this regard, a preliminary report involving a six month, double-blind placebo-controlled study of 28 patients split into two parallel groups is of interest; it suggests that the anti-inflammatory agent indomethacin slows cognitive deterioration compared to placebo in patients with dementia of Alzheimer's type. This effect is postulated to involve immune reactions in plaque formation. Although intriguing, replication studies are needed with larger sample sizes and different anti-inflammatory agents.

## FUTURE DIRECTIONS

Recently, researchers have found a strong association between the most common form of Alzheimer's disease (i.e., the late onset type beginning after age 60 years) and an abnormal variant of the apolipoprotein - E gene on chromosome 19.<sup>11</sup> People with two apoE4 genes have eight times the risk of developing late-onset Alzheimer's disease as those with two of the more common apoE3 genes. A person with one apoE4 gene has a greater than 40 percent risk of developing Alzheimer's disease by age 80 years, and a person with two apoE4 genes has a greater than 90 percent risk of developing Alzheimer's disease by age 80 years. The apoE4 gene is not the final answer because Alzheimer's disease does not have a single cause. Rare familial forms of early onset Alzheimer's disease have been linked to chromosomes 14 and 21. ApoE4 may contribute to as much as 75 percent of the late-onset form of Alzheimer's disease, but much of the variance remains unexplained and other risk factors must play a role. Further, the relationship of the apoE4 gene to the pathological processes is unknown, and ApoE4 has been related to other dementias (e.g., vascular). However, this is an important breakthrough because it offers the opportunity to investigate the mechanisms by which apoE4 leads to an increase risk of Alzheimer's disease. Understanding these mechanisms provides the best hope for developing therapeutic strategies with dramatic efficacy. □

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# SCMA NEWSLETTER

A PUBLICATION OF THE SOUTH CAROLINA MEDICAL ASSOCIATION  
Joy Drennen, Editor  
798-6207, in Columbia

Contributions welcomed  
1-800-327-1021, outside Columbia

November 1994

## MEDICARE UPDATE

By now you should have received the November *Medicare Advisory* which you should read very carefully.

**EPO for Non-ESRD Patients:** HCFA has established a new code effective 11/14/94 to report EPO administered to non-end stage renal disease patients. The code is Q0136.

Participating Fee Amount:	\$12.00
Non-Participating Fee Amount	\$11.40
Limiting Charge:	\$13.11

**Interest Rate Update:** Effective 10/4/94, the interest rate for overpayments and underpayments is 13.375 percent. This applies to amounts due from a provider when a claim has been overpaid, and also to the amount underpaid by Medicare when additional benefits resulting from an appeal or hearing are not paid within 30 days of determination.

**Corrected Claims:** If you discover an error on a claim that has already been paid, you must send a corrected claim hard copy. Corrections or changes should be circled in red and "corrected claim" should be written at the top in red.

### **Vaccine Reminder:**

<u>Vaccine Code</u>	<u>Administration Code</u>	
	<u>On or After 10/1/94</u>	<u>Prior to 10/1/94</u>
Flu-90724	G0008	Q0124
Pneumococcal-90732	G0009	Q0124
Hepatitis B-90731	G0010	W0125

Flu and pneumococcal injections are not subject to non-participating reductions and limiting charge guidelines. Medicare pays 100 percent of fee schedule amount. Charges do not apply against deductible or coinsurance. Hepatitis B vaccines are subject to non-par reduction and limiting charge guidelines. These charges may be applied to deductible and coinsurance.

**Telephone Numbers Changing:** Effective December 5, 1994, Medicare Part B extension numbers (for anyone located in the Alpine Tower) will have a "4" added to the beginning of their four-digit extension number. Extension numbers of Part A offices in Camden will begin with "2."

**New Address:** The new mailing address for the Hearings Department is Medicare Part B Hearings, PO Box 100249, Columbia, SC 29202. ☐

## CAPSULES

Robert H. Taylor, MD, of Spartanburg, has been recognized by the American Academy of Family Physicians for his longtime support of the medical specialty of family practice. Dr. Taylor received the AAFP's Award of Merit, which recognizes significant contributions to the advancement of family practice and the AAFP.



**Reality Condom:** The new Reality female condoms are Medicaid reimbursable through the prescription program. Any prescriptions classified as family planning do not count against the limit of three prescriptions a month for Medicaid recipients.

**Family Planning:** As a reminder, the family planning waiver was implemented on July 1, 1994. The waiver provides family planning coverage for an additional 22 months to women who would normally lose their Medicaid eligibility at the end of the post-partum period. The waiver provides coverage to women for **services or complications directly related to family planning only**. The eligibility cards for the family planning waiver are gold plastic cards which closely resemble a credit card. At the top of this card you will see the following statement: "South Carolina Medicaid Card for Family Planning Prescriptions and Services Only." An example of the card and further details can be found in Section 209.1a, on page 200-

54.1, of the Physicians, Laboratories and Other Medical Professionals Medicaid Provider Manual.

**1995 CPT Codes:** The 1995 codes will be available for billing January 1, 1995. A bulletin will be forthcoming with policy and system changes related to the new codes.

**Changes in NICU Codes:** The Health Care Financing Administration has decided that the Health and Human Services Finance Commission is not in violation of federal regulation by using CPT codes and instructions for neonatal intensive care unit services. Effective sometime in 1995, the Neonatal Intensive Care CPT codes will replace the current codes covering neonatology services.

*For more information, please contact Elizabeth Biggers at (803) 798-6207, ext. 236 or (800) 327-1021, ext. 236.* □

## MEDICAID WAIVER UPDATE

The Health Care Financing Administration (HCFA) has approved in concept South Carolina's 1115 Medicaid waiver entitled The Palmetto Health Initiative. HCFA still has concerns regarding the proposed managed care program, but has assembled a team to address the problems. The Health and Human Services Finance Commission lists the following issues as "key milestones" which must be resolved before HCFA will grant final approval of the waiver:

1. Develop a contract for the Member, Access and Choice Coordinator (MACC)
2. Finalize the capitated rates
3. Demonstrate adequacy of service delivery
4. Define the requirements for the partially capitated program
5. Adapt the Medicaid Management Information System
6. Create a quality assurance plan for the fully and partially capitated programs
7. Assure budget neutrality
8. Define the Long Term Care Pilot's processes
9. Develop an implementation plan with stated milestones

The Finance Commission will begin the program approximately one year from the final-approval letter.

The SCMA will soon implement a Medicaid Waiver Hotline for members to call with questions and comments regarding the Palmetto Health Initiative. Please look for the announcement in the December or January newsletter.

## PHYSICIANS CARE NETWORK UPDATE

Four more hospitals have contracted with Physicians Care Network: Charter Hospitals of Augusta, Charleston, Columbia and Greenville. This brings the total number of hospitals to 29.

Approximately 2,600 physicians have enrolled in the network to date.

*For more information regarding PCN, please call Barbara Whittaker, in Columbia at 798-6207, ext. 226 or statewide at 1-800-327-1021, ext. 226.*

## AMA HOSPITAL MEDICAL STAFF SECTION 24TH ASSEMBLY MEETING

Send a representative from your hospital medical staff and physician organization to the 1994 Interim AMA Hospital Medical Staff Assembly Meeting to be held December 1-5, 1994 at the Sheraton Waikiki Hotel in Honolulu, Hawaii. Aside from participating in the development of AMA policy, representatives will have an opportunity to network with colleagues, dialogue with the AMA Board of Trustees, and hear the latest news and information on health system reform.

*For information, contact Michael R. Vitek, AMA Department of Hospital Medical Staff Services, (312) 464-5000.*

## REMEMBER STARK II

Effective January 1, 1995, a new federal law called STARK II prohibits physicians with an ownership or investment interest in, or a compensation agreement with, an entity from referring Medicare and Medicaid patients to that entity for the following health services: clinical laboratory services (set forth in STARK I, 1990); physical therapy services; occupational therapy services; radiology or other diagnostic services; radiation therapy services; the furnishing of durable medical equipment; parenteral and enteral nutrients, equipment and supplies; prosthetics, orthotics and prosthetic devices; home health services; outpatient prescription drugs; and inpatient and outpatient hospital services.

STARK II restricts group practices from distributing revenues or compensating physicians based on the volume or value of referrals. Violators are subject to fines from \$15,000 to \$100,000.

**Physicians are strongly urged to review their referral arrangements, investments and compensation formulas.**

*For further information, please contact Elizabeth Biggers in Columbia at 1-803-798-6207, ext. 236, or statewide at 1-800-327-1021, ext. 236.* ☐

## AMA LEADERSHIP CONFERENCE GRANTS AVAILABLE

The AMA Medical Student Section and Resident Physicians Section are pleased to announce the second annual AMA/Glaxo Achievement Award Program. Twenty-five resident physicians and 25 medical students will receive funding to attend the 1995 AMA National Leadership Conference April 22-26, 1995.

Made possible through a grant from Glaxo, Inc., this program will recognize medical students and residents who have exhibited outstanding leadership abilities in organized medicine, civic or non-clinical medical school or hospital activities. Winners will be able to augment their leadership abilities through participation in this annual conference which features a prominent faculty of nationally recognized members of the government, media and the private sector. A special session specifically designed for residents and students will concentrate on improving overall leadership/communication skills.

*Students and residents interested in applying for this AMA/Glaxo Achievement Award should contact 1-800-AMA-3211 to receive an application. The application postmark deadline is December 16, 1994.* ☐

I would like to welcome you to the first of many newsletter articles from the Young Physicians Section (YPS). The YPS consists of all physicians under the age of 40 or in their first five years of practice. Currently there are approximately 1,200 such physicians in South Carolina. The YPS is active and growing and would welcome any members who would be interested in becoming more involved.

The mission of the Section is to provide a means for young practicing physicians to address and subsequently refer common problems, issues and/or interests to the SCMA Board of Trustees and House of Delegates. As such, the YPS Governing Council is charged with educating new physicians and physicians in training about the activities of the AMA and SCMA, and the importance of personal involvement in organized medicine. Several activities by YPS leaders have aided young physicians in learning the intricacies of managed care, contractual relationships, and the Physicians Care Network. The current goals for the YPS Council are as follows:

- Coordinate a series of regional conferences entitled, "Survival Strategies Under Managed Care/Capitation." A brand new text on contract negotiation produced by the Young Physicians Section and the AMA will be utilized in these conferences. They are planned for early 1995; stay tuned...
- Improve legislative awareness and involvement by developing a statewide network of legislative key con-

tacts. You will receive further information after the November elections.

- The appointment of a member of the YPS to the Editorial Board of the SCMA journal. This position has already been approved by *The Journal* Editorial Board and the YPS Council is currently seeking interested YPS members. Interested members should contact Cathy Boland at the SCMA Headquarters.
- Lastly, in an effort to improve communication with Section members, periodic editorials or articles will be included in the SCMA journal dealing with various issues relevant to those of us who thus far are naive about the business and/or politics of medicine. Various public health issues will also be discussed with an emphasis on specific ways in which we can help prevent needless illness or injury in our patients.

These goals, however lofty, were chosen to increase the fund of knowledge and activity level of the young physicians in South Carolina. We need to realize that any health care system reform or insurance measure will affect us to a greater degree solely due to our duration of practice. I am confident that the more active the physicians of South Carolina are in future reform discussions, the brighter the future will be for our patients and ourselves. □

Richard A. Schmitt, MD  
Chairman, YPS

### WORKSHOP SCHEDULED

A workshop entitled "Protecting your Medical Practice II" will be held on Saturday, December 10, 1994, 8:30 am–5:30 pm, at the University of South Carolina School of Medicine in Columbia, Medical Library Building, Room 301. The topic will be Continuing Medical Education in Alcohol and Drug Issues for Physicians. The workshop has been approved by the Physicians Advocacy and Assistance Committee, the SCMA, and the USC School of Medicine.

The speakers include Doctors Ted Watson, Craig Davis, William Green, Kathleen Brady and N. Peter Johnson; Wilbur Harling of DHEC Drug Control; Henry Foster of the State Board of Medical Examiners; and Vic Pascal of SCMA Financial Services.

Address all inquiries to the co-chairmen, N. Peter Johnson, PhD (803) 733-3191, or Martin Zwerling, MD, (803) 648-9555. □



# NEW APPROACHES IN THE TREATMENT OF ALZHEIMER'S DISEASE\*

JACOBO E. MINTZER, M. D.\*\*  
DAVID BACHMAN, M. D.

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Alzheimer's Disease (AD) is a disorder characterized by a slow and progressive process of deterioration in cognitive functions. Cognitive deterioration occurs in areas of memory, abstract thinking, judgment, insight, language, comprehension and constructional abilities. As the disease progresses, personality changes are also likely to occur, as well as deterioration in functional abilities.<sup>1</sup> As a result of this devastating process of deterioration, patients often develop a variety of behavioral disturbances, such as paranoia, depression and agitation.<sup>2</sup> Although a cure has yet to be discovered for AD, various treatments are available to limit its symptoms and behavioral disturbances, to decrease the suffering experienced by AD patients and their families.<sup>3</sup> Currently, most clinicians agree that treatment of AD patients should focus on three major areas: (1) improvement of cognitive functions by slowing and/or stopping the progression of the disease, (2) managing or preventing behavioral disturbances, and (3) providing support and guidance to the caregiver.<sup>3</sup> This review briefly summarizes available information in each of these areas.

## IMPROVEMENT OF COGNITIVE FUNCTIONS BY SLOWING AND/OR STOPPING THE PROGRESSION OF THE DISEASE

AD appears to have many possible causes.

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However, despite multiple etiologies, the pathogenesis of this disorder appears to involve a single common pathway which gives the illness its characteristic histologic and clinical features. The accumulation of beta amyloid with subsequent development of senile plaques may be the unifying cause of this disorder.<sup>4</sup> In the last five to 10 years, epidemiologic and molecular genetic studies have suggested several mechanisms that may result in the accumulation of beta amyloid in the brain. Fifty or more families have been described in which Alzheimer's disease occurs as an autosomal dominantly inherited disorder. For most of these families, the illness has an early onset, in the 40s or 50s. In about 15 percent of these families, specific base pair substitutions have been identified in the amyloid precursor protein (APP) gene on chromosome 21.<sup>5</sup> Beta amyloid is a fragment of the larger APP, and the mutations identified a bracket of the segment of the APP gene which codes for the beta amyloid fragment. In those prone to develop AD, it is very likely that when APP is cleaved after incorporation into the neuronal membrane, subsequent metabolism results in the formation of the relatively indigestible beta amyloid fragment rather than a soluble APP fragment. Gradual accumulation of beta amyloid by this process may result in eventual plaque formation and neuronal death.

For most families with early onset familial Alzheimer's disease (FAD) in whom chromosomal analysis has been performed, a specific genetic mutation has not yet been identified. However, using indirect chromosomal analysis, a specific section of chromosome 14 has been identified.<sup>6</sup> The nearest marker identified on chromosome 14 to date, D14S43,

shows little recombination with FAD, suggesting that the marker is part of the gene complex or very near it. It is expected that the gene product will likely be involved in the expression or metabolism of APP or beta amyloid, but this is unproven at present.

Families with late onset FAD demonstrate disease association with a marker on chromosome 19.<sup>7</sup> This collection of families is particularly important, since late disease onset (after age 60) is typical of the more common sporadic form of AD. Although the specific target gene on chromosome 19 is not yet known with certainty, linkage is close to the apolipoprotein E (APOE) gene. APOE occurs in three isoforms (e-2, e-3, and e-4), with e-3 being the most common isoform in most populations. Corder et al.<sup>8</sup> found that risk for AD increased from 20 to 90 percent with an increasing number of alleles for e-4 in 42 families with late onset AD. Other research centers have also confirmed an association between APOE e-4 and late-onset AD. The role that APOE e-4 plays in the development of AD is not known. It appears that e-4 may bind to beta amyloid more than other APOE isoforms. Alternatively, it has been suggested that a paucity of e-3 rather than an excess of e-4 is the critical problem. Isoform e-3 appears to bind to neurotubulin, an important element of the axonal tubular system, preventing the degradation of neurotubulin into neurofibrillary tangles. Further research is necessary to determine whether APOE is involved in the fundamental pathogenesis of AD, or simply a biological marker for some and not others. Should it be discovered that a shortage of APOE e-3 contributes significantly to the pathogenesis of AD, researchers suggest that the development of treatments attempting to increase APOE e-3 availability will be the focus of AD research activity in the near future.

Until the underlying cause of AD is established in a majority of cases, a definitive therapy will not be available. Preventive therapies may be directed at halting the accumulation of beta amyloid in the brain, or at facili-

tating the disposition of beta amyloid in the brain. A number of alternative preventive therapies are under study. Limited epidemiological data suggest that postmenopausal women who take estrogen replacement therapy may be at somewhat lower risk for developing AD than women who do not. Estrogen, when combined with nerve growth factor, appears to have a protective effect on cholinergic neurons.<sup>9</sup> Clinical trials are under way. The ultimate deposition of beta amyloid in the brain may require the activity of certain complement factors. It has been hypothesized that anti-inflammatory drugs that reduce complement production may slow the development of AD. A recent twin study suggested that anti-inflammatory drugs, especially ACTH or steroids, may postpone or prevent the development of AD.<sup>10</sup> Clinical trials with indomethacin are in progress. Clinical trials are also under way using nerve growth factor stimulating compounds, selegiline to prevent the formation of free radicals, and calcium channel blockers as well as other drugs.

Symptomatic treatment of Alzheimer's disease is better established. In August 1993, the FDA approved marketing of tacrine, the first drug in the United States specifically for the treatment of the cognitive symptoms of AD.<sup>11</sup> An acetylcholinesterase inhibitor, tacrine has proven to be a problematic drug. Although it demonstrates some efficacy in 20 to 40 percent of patients with early to middle stage AD, tacrine produces elevation of liver enzymes (particularly ALT levels) in approximately 50 percent of cases. Liver enzyme elevations are reversible (and generally asymptomatic) but require weekly monitoring of ALT levels until the patient is well established on the highest possible dose of the medication. In our experience, tacrine can be a very effective drug, despite its serious limitations. We have been particularly impressed that while patients may demonstrate improvement in communication, initiative and sociability, improvement in memory may be more limited. Other drugs which either enhance acetylcholine function or multiple neurotransmitter

systems are also under study.

## MANAGING OR PREVENTING BEHAVIORAL DISTURBANCES

In addition to their cognitive difficulties, demented patients present with significant behavioral disturbances such as depression, psychosis and agitation. The most prevalent behavioral disturbance is agitation. Agitation is defined as "inappropriate behavior unrelated to unmet needs or confusion."<sup>12</sup> Symptoms of agitation include wandering, hitting, kicking, grabbing, screaming, intentional falling, performing repetitious acts or causing injury to self or others. Over 60 percent of demented patients develop agitation at some point during the course of their disease.<sup>13</sup> We call this type *primary* agitation. Secondary to AD, multiple medical and psychiatric disorders such as depression and psychotic disorders can also cause agitation. If secondary disorders are diagnosed and treated, this type of agitation may be reversible.<sup>2</sup>

## AGITATION IN AD

Currently, primary agitation associated with AD is treated symptomatically with pharmacological agents such as low dose neuroleptics, anti-convulsants such as carbamazepine and valproate, and anxiolytics such as short acting benzodiazepines and buspirone.<sup>14,15</sup> This approach is not beneficial to all patients with AD and agitation.<sup>16</sup> Caregivers often lack ability to manage symptoms of agitation in the home environment, increasing the suffering of patients and their caregivers, with increased risk of institutionalization.<sup>17-20</sup>

### *Treatment Alternatives*

An alternative/complementary approach to pharmacotherapy in the treatment of elderly demented patients with severe agitated behaviors is the use of behavioral/environmental techniques.

### Theoretical Background for a Behavioral Environmental Approach

*Excess Disability:* Excess disability is

defined as "degree of functional impairment in excess of that expected, due to the level of intellectual impairment caused by the dementing process."<sup>21</sup> Effective treatment in institutionalized demented elderly patients depends on identifying sources of excess disability. When adequate attention is devoted to the elements causing excess disability, clear potential for improvement is observed. A common source of excess disability is the presence of medical and psychiatric disorders that manifest only as agitation. Identification and treatment of medical or psychiatric disorders that manifest as agitated behaviors will reduce agitation and its negative consequences, such as nursing home placement.<sup>21</sup>

Unfortunately, most demented patients cannot aid the clinician in discovering the source of disability due to language impairment, lack of memory, or difficulties in processing information. Discovery relies on the availability of a highly controlled environment; through a process of systematic elimination, the possible sources of excess disability are ruled out.

*Catastrophic Reaction:* Another important concept is Kurt Goldstein's "catastrophic reaction,"<sup>22</sup> suggesting that violent reactions observed in post-stroke patients are often related to environmental demands that exceed the patient's functional abilities, e.g., encouraging conversation in a patient suffering from a language impairment. The same principles apply to demented patients. In our experience, when stimulated or exposed to levels of demand which exceed their functional abilities, demented patients respond with agitated behavior. The management of the patient in an overstimulating or overdemanding environment (in relation to the patient's abilities) will *trigger* the onset of a catastrophic reaction that manifests as agitated behavior. The continued presence of these elements in the patient's environment will *sustain* these behaviors, and contribute to further deterioration, especially if the environmental triggers of these behaviors remain unidentified. The need to avoid the development of catastrophic



reaction-type behaviors has been generally acknowledged.<sup>23-26</sup> Experts emphasize the need for understanding the characteristics of the patient's behaviors, for caregiver education, for environmental modification, and for flexibility in the approach.

*Adaptation of the Home Environment:* The home environment is frequently very stimulating for AD patients (TV, lights, furniture). A non-trained caregiver can provoke catastrophic reactions by demanding a level of performance identical to the one the AD patient once had, before the development of the dementing process. An environmental approach calls for training the caregiver regarding the patient's limitations and eliminating overstimulating elements in the home environment. Further, the patient and family lifestyle can be modified to meet the patient's needs.

The impact of these treatment principles has been demonstrated at the MUSC Behavioral Intensive Care Unit (BICU), which specializes in the treatment of behavioral disturbances, particularly severe agitation associated with AD.

The BICU approach to the treatment of severe agitation in cognitively impaired patients is based on the assumption that agitated behaviors are not random, but are largely determined by undetected medical problems (such as asymptomatic urinary tract infections or pneumonia) or by environmental elements that *trigger, sustain* or *extinguish* the observed behavior.<sup>27</sup>

The BICU model consists of the following components.<sup>28</sup>

1. Identification of agitated behaviors: In a highly controlled environment, agitated behaviors are identified through intensive observation.
2. Extensive multidisciplinary evaluation: An extensive multidisciplinary evaluation is conducted in order to identify factors responsible for the agitated behavior, i.e., medical/psychiatric, environmental, and/or social factors.
3. Design and implementation of the treat-

ment strategy: Behavioral interventions are developed and implemented to address those factors found to contribute to the patient's agitation. If no medical or psychiatric factors can be found to explain the agitation, pharmacotherapy is utilized to minimize the agonizing symptoms of agitation.

4. Caregiver and environmental interventions: Caregiver and environmental interventions include primary caregiver training and home modification. Primary caregivers are taught how to reduce the patient's agitated behavior through various behavioral management techniques. Home modifications are implemented to adapt the home environment to the specific needs of the AD patient and to increase the patient's ability to adapt to demands of the family and home environment.<sup>27</sup>

An evaluation of BICU treatment outcomes shows that a comprehensive multidisciplinary assessment, combined with treatment implementation and caregiver and environmental intervention appears to be effective in decreasing agitation in AD patients. A small group of patients (20 percent) treated in the BICU shows complete resolution of the symptoms of agitation; the majority (66 percent) of demented agitated patients show decreased agitation.<sup>28</sup> Based on these outcomes, the application of these treatment principles has a positive impact in the care of AD patients.

## OTHER PSYCHIATRIC DISORDERS

AD patients often present with psychiatric disorders such as depression (40 percent) and/or psychotic disorders (40 percent); either separately or concurrently with agitation. A specific treatment approach appears to decrease agitation and to have a positive impact on the AD patient's well-being. Although the scientific literature does not clearly address specific dosing, maintenance, or treatment protocols used to manage AD patients suffering from psychiatric disorders,

a broad range of clinical experience is available. From clinical experience, we believe the most effective approach to the management of psychiatric disorders in AD patients is as follows:

(1) For AD patients presenting with psychotic symptoms (especially paranoid delusional symptoms), the first approach is to utilize neuroleptic medications such as haloperidol at doses of approximately 0.03 mg/Kg.<sup>29</sup> New antipsychotic medications such as clozapine are also used to treat AD patients with psychotic symptoms. Some patients show a clearly positive response with minimal side effects, while others develop severe side effects.<sup>30</sup> When using clozapine, we recommend starting at daily doses of 6.25 mg and titrating up as clinically indicated in an inpatient setting is recommended.

(2) For AD patients suffering from major depression, tricyclic antidepressants with mild anticholinergic and orthostatic effects are useful. Medications with strongly anticholinergic side effects and those which produce orthostatic hypotension should be avoided if possible.<sup>31</sup>

Selective serotonin re-uptake inhibitors (SSRI) can also be useful in treating AD patients with major depression; however, all antidepressants must be used with caution. Certain antidepressants (including SSRI) can cause a syndrome of inappropriate secretion of antidiuretic hormone (SIADH) which may lead to harmful complications.<sup>32</sup> Medications like desipramine and nortriptyline, used at low starting doses and carefully titrated, are important therapeutic aids in managing depressed AD patients.<sup>31</sup> There is evidence to suggest that elderly AD patients presenting with major depression and multiple medical problems can benefit from treatment with stimulants such as methylphenidate.<sup>33</sup>

Researchers and clinicians participating in the National Institute of Mental Health Consensus Conference on Depression in the Late Life agreed on the importance of using aggressive pharmacotherapy.<sup>34</sup> Electro-convulsive Therapy (ECT) may be useful in the treat-

ment of demented elderly patients who have psychotic symptoms with major depression.<sup>35</sup> For more on the use of ECT in demented elderly patients, refer to the Textbook of Geriatric Neuropsychiatry.<sup>36</sup>

## PROVIDING SUPPORT AND GUIDANCE TO THE CAREGIVER

Spouses and adult children of patients with AD provide the majority of supportive health and social services, including assistance with essential activities such as bathing, feeding, dressing and toileting.<sup>37</sup> They also coordinate medical and community services for the demented family member.<sup>38</sup> These duties impact significantly on the caregiver's daily routine.<sup>39</sup> About half of the caregivers manage well; the remaining half suffer from associated mental health and medical problems.<sup>17,40-43</sup>

Caregiver burden in coping with an AD patient's problematic behavior at home, especially agitation, strongly correlates with the decision to institutionalize an AD patient.<sup>17,20</sup> We believe that support groups, case management services and respite programs are essential components of AD treatment. Caregiver support and training are widely accepted as one of the few measures that will impact the quality of life for the AD patient and the caregiver, as well as the caregiver's mental health problems.

## CONCLUSION

Years of research and clinical work are finally starting to pay off. Although a definite cure for Alzheimer's Disease has yet to be discovered, clinicians currently have treatments available to alleviate or to minimize the behavioral disturbances that occur in AD, and for some individuals, to actually improve cognitive function. These advances are greatly decreasing the suffering experienced by AD patients and their families. We believe that the future will be even brighter, if researchers, clinicians, patients, family members and the community at large continue to merge their efforts in the battle against this devastating disease. □

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# ALUMINUM NEUROTOXICITY AND ALZHEIMER'S DISEASE

CHARLES N. STILL, M. D.\*

Controversy continues concerning the nature of the association between aluminum neurotoxicity and Alzheimer's disease (AD). A fundamental conflict among investigators regarding the role of aluminum in AD involves timing and causality, i.e. whether aluminum deposition is a cause of AD or a result of AD.<sup>1</sup> This review focuses on toxic exposure to aluminum compounds and the subsequent occurrence of dementia, with emphasis on possible protective effects of fluoride against aluminum neurotoxicity and the development of AD in late life.<sup>2</sup>

Aluminum is the most abundant metallic element. Earth's surface is composed of about eight percent aluminum by weight, principally laterite and bauxite, often combined with aluminum silicate. Aluminum sulfate (alum) is also plentiful, and it has been used as an astringent and mordant since the 5th century BC. Alum also serves as a flocculant in water purification. Public water supplies usually contain about 1 ppm of aluminum. Since most aluminum compounds are usually insoluble at pH ranges from 6.0-8.0, animals and plants historically have carried low tissue burdens of aluminum. However, acid rain and fertilizers have leached additional aluminum from rocks and soil into surface water reservoirs, thereby creating new hazards of aluminum toxicity for plants and animals, including man. Pharmaceutical progress has also provided a variety of aluminum-containing medications which can be administered parenterally or orally. The kidney offers the only known route for removal of aluminum from the body; impaired renal function is associated with marked increases in body and brain aluminum levels, especially from par-

enteral aluminum administration. Aluminum clearance occurs at about five to 10 percent of the glomerular filtration rate. Whether oral aluminum intake can overload the normal kidney is uncertain.<sup>3</sup>

## DIALYSIS DEMENTIA AND AD

Impaired renal function is associated with marked increases in the total body burden of aluminum after oral loading, leading to aluminum encephalopathy, also known as dialysis dementia.<sup>3</sup> Aluminum neurotoxicity was first reported in 1885 by Siem, who showed that repetitive injections of sodium aluminum tartrate produced progressive neurological impairment in dogs, consisting of apathy, myoclonus, medullary and spinal paralysis, convulsions and death. Siem concluded that the central nervous system is the critical point of attack in systemic aluminum poisoning.<sup>4</sup> Dialysis dementia was first reported in 1972 by Alfrey et. al. in patients on long-term hemodialysis for chronic renal failure. Before clinical onset, electroencephalograms showed abnormal patterns of excessively slow background activity and occasional spike and wave complexes. The clinical course was characterized by apraxia, asterixis, tremor, myoclonus, memory impairment, personality changes, loss of speech, motor incoordination, seizures and death within six to nine months after onset. Aluminum levels in cerebrospinal fluid and in cerebral cortex were markedly elevated. Specific neuropathologic changes were not found; subsequent studies showed that the occurrence of dialysis dementia was correlated with high concentrations of aluminum in the dialysis water, as well as the total duration of dialysis. When water deionization was used to maintain the aluminum concentration of dialysis water below 10 mcg/l, the frequency of dialysis

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dementia fell abruptly, recurring when aluminum levels exceeded the recommended limits.<sup>4</sup> Moreover, short-term experimental use of desferrioxamine (DFO) during dialysis substantially reduced plasma aluminum levels, with marked cognitive improvement. Long-term effects of chelation therapy for dialysis dementia are still unknown.<sup>5</sup>

McLachlan et. al. have reported a two-year randomized single-blind placebo-controlled clinical trial of DFO in 48 AD patients, using intramuscular injection to give 125 mg twice daily on five days each week. Both the treatment group and the non-treatment group were closely matched at the outset of the study; the nontreatment group deteriorated at twice the rate of the DFO treatment group, based on videotaped home behavioral assessments. Trace-metal analysis of brain tissue taken at autopsy showed that DFO treatment reduced neocortical aluminum concentrations to near the control values.<sup>6</sup>

### BLOOD-BRAIN BARRIER

Experimental animal studies have shown that the blood-brain barrier is protective against aluminum neurotoxicity. In rats, intraperitoneal injection of aluminum chloride reversibly altered blood-brain barrier permeability, with marked deposition of aluminum in brain lysosomes, followed by lysosomal degeneration and cell death. Aluminum levels were lower in the nuclear fractions of rat brain in exposed rats than in controls, suggesting that the aluminum became sequestered in other cellular compartments before reaching the nucleus.

Bypassing the blood-brain barrier gave remarkably different results. Within hours after intracerebral and/or intraventricular injection of aluminum salts in cats, dogs, or rabbits, aluminum localized in the nucleus, binding to nuclear chromatin, accompanied by clinical evidence of impaired memory and defective learning, suggesting that aluminum may produce adverse effects on information transcription before neurofibrillary changes develop. In aluminum-treated rabbits, neu-

rofibrillary tangles (NFT) were composed of 10 nm-diameter filaments, unlike the paired helical filaments with a periodicity of 80 nm in AD. Human cortical neurons also develop neurofibrillary changes in tissue culture when exposed to aluminum. Compounds of other metallic elements have not been shown to have such effects.<sup>5</sup>

### ALZHEIMER'S CASE

In 1907 Alois Alzheimer reported the clinical and neuropathologic findings in the case of a German housewife who died at age 55 of a relentlessly progressive illness lasting four and a half years. Her illness began with jealousy about her husband, swiftly followed by memory impairment, disorientation to time and place, paranoid ideation and behavior, and auditory hallucinations. Though she could still walk and use her hands normally, she showed severe impairment in speaking, reading, writing, and understanding questions. She could not name objects or describe their proper uses. At the end stage of her illness, she was totally apathetic, incontinent, and confined to bed in a fetal position, with her legs drawn up. Despite good nursing care, she developed decubitus ulcers. The autopsy showed brain atrophy with arteriosclerosis of the larger cerebral blood vessels. Silver staining of the cerebral cortex revealed NFT which replaced the nucleus and cytoplasm of affected neurons.

Alzheimer noted that NFT stained with dyes differently from normal neurofibrils; he postulated chemical transformation, with deposition of an unidentified pathological product in neuritic plaques. Glial cells also showed overabundant fibers and large deposits which could be microscopically identified even when unstained. He concluded that this was a new disease which could not properly be included among previously recognized mental diseases.<sup>7</sup>

AD was known as a form of presenile dementia until 1964, when Kidd and Terry independently showed that AD and senile dementia of the Alzheimer type share identi-



cal neuropathological features at the ultra-structural level. From a prospective study of elderly persons, Blessed et. al. found a strong correlation between mental status-functional ability and the number of neuritic plaques in the cerebral cortex at death. The degree of cerebral arteriosclerosis was not specifically related to the presence or absence of dementia. AD is strongly age-associated, with the highest incidence in the ninth decade. The impact of these studies ultimately led to the recognition of AD as a major public health problem affecting four million Americans.<sup>8</sup>

In 1980, the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders introduced a new synonym for AD, called primary degenerative dementia (PDD). Diagnostic criteria were based on clinical features of intellectual deterioration of sufficient severity to interfere with social or occupational functioning, associated with impairment of memory, judgment, and higher cortical functioning in clear consciousness. After autopsy studies revealed that 20 percent of those clinically diagnosed with AD actually had other diseases, the Department of Health and Human Services Task Force on Alzheimer's Disease further refined the diagnostic criteria into Probable, Possible, and Definite categories of AD, emphasizing the importance of documenting the absence of systemic disorders or other brain diseases which could account for progressive deficits in higher cortical functioning.<sup>9</sup>

### BRAIN ALUMINUM

Brain aluminum concentration increases with normal aging. A Canadian study reported high concentrations of aluminum in AD brain, attributed to the use of aluminum to purify drinking water in Canada. Other investigators found similar increases in brain aluminum, but without significant differences between AD patients and controls.<sup>8</sup> Aluminum is located in NFT, and aluminosilicates have been identified in the core of neuritic plaques in AD brain tissue. Experimental evidence suggests that defects in the olfactory

bulbs and/or the blood-brain barrier allow aluminum influx into the brain; the mechanisms underlying these complex neuropathologic changes remain unknown.<sup>8,10</sup> Laser microprobe mass analysis (LAMMA) of NFT-bearing and NFT-free hippocampal neurons showed selective accumulation of aluminum in the NFT of 10 AD patients.<sup>11</sup>

### ALUMINUM IN WATER

Epidemiologic studies in 88 county districts of England and Wales have shown that estimated rates of AD in persons under age 70 can be correlated with mean concentrations of aluminum in drinking water during the prior decade. These studies involved 1,185 patients with dementia and 2,920 patients with epilepsy. The diagnosis of AD was based on standardized clinical criteria, with CT scans reported as normal or showing only cerebral atrophy. If water aluminum concentrations (WAC) were less than 0.01 mg/l, rates for probable AD were 5.4 per 100,000; for possible AD, 3.0 per 100,000; and for other types of dementia, 6.3 per 100,000. The relative risk of AD where the WAC was higher than 0.11 mg/l was 1.5 times greater than where the WAC was lower than 0.01 mg/l. There was no evidence of a relationship between the WAC and epilepsy or other types of dementia.<sup>12</sup>

### ALUMINUM FLUORIDE COMPLEXES

In 1936, Sharpless did studies in rats showing that diets containing sodium fluoride (1000 ppm) retarded growth. If aluminum chloride (0.056 percent) was also added to the diet, growth was normal. Moreover, aluminum chloride (two percent) retarded dental fluorosis for six months. Sharpless noted that aluminum combined with fluoride to form a compound which was not absorbed from the gut. Subsequent investigations confirmed that aluminum sulfate (0.53 percent) fed to young rats on high sodium fluoride (445 ppm) diets resulted in a 45 percent reduction of bone fluoride content. Dental fluorosis also occurred in cattle and sheep with fluoride intakes above 5 ppm. With fluoride intakes around 98

ppm, they developed bone and joint lesions leading to lameness in about a month.<sup>13</sup> Aluminum salts were found to be effective "fluoride alleviators" by decreasing fluoride absorption from the gastrointestinal tract.<sup>13</sup> Brudevold et al. analyzed for total and complexed fluorides in tap water from 26 communities in Colorado, Massachusetts and North Dakota. Complexed fluorides were highest in waters treated with aluminum sulfate. Of 14 trace elements usually found in drinking water, only aluminum produced fluoride complexation within the usual pH ranges. Calculations showed that fluoride complexation may occur over the entire pH range of the stomach and the duodenum. Does long-term fluoride administration effectively decrease the absorption of aluminum in man? This problem deserves further study.

### FLUORIDE IN WATER

In South Carolina, water fluoride content (WFC) exceeds maximum contaminant levels in many localities of the Atlantic seaboard. Still and Kelley<sup>2</sup> completed the only epidemiologic study using WFC as the protective exposure factor against aluminum neurotoxicity as a risk factor in the development of primary degenerative dementia (PDD).

In this retrospective cohort study, residents of Horry County (WFC 4.18 mg/l), York County (WFC 0.61 mg/l), and Anderson County (WFC 0.49 mg/l) were identified by continuous WFC exposure for at least 10 years in the specified county of residence. Case records of the South Carolina Department of Mental Health were examined for evidence of PDD and other types of dementia. Patients aged 55 or older at first admission were identified during the eight year period beginning July 1, 1971 and ending June 30, 1979. Cases were classified by DSM-III criteria, and tabulated by diagnosis, race, and sex. Annual incidence rates per 100,000 per year were calculated. The 2 x 2 chi-square distributions revealed significant differences only for Horry County's PDD cases, which showed an incidence of 3.6/

100,000, versus 17.1/100,000 for York County and 20.8/100,000 for Anderson County. These rates were not corrected for age; the study did not differentiate between ground water and surface water sources, which may differ in WFC.

Since 1969, municipalities in Anderson and York Counties have had artificially fluoridated water supplies (WFC 1 ppm). The U. S. Environmental Protection Agency's maximum contaminant levels (MCL) were adopted in May 1986: the primary MCL is 4.0 ppm, and the secondary MCL is 2.0 to 3.9 ppm. In June 1988, Myrtle Beach, the largest city in Horry County, opened its own municipal surface water treatment plant, with an adjusted WFC of 1 ppm, with an estimated WAC contamination of 1-3 ppm due to aluminum sulfate, which is used as a flocculant. The remainder of Horry County still depends on ground water supplies which equal or exceed the MCL established by the EPA. Based on 1985 population estimates for South Carolina, 82.4 percent of the population is supplied by public water systems (PWS), with 63.5 percent having PWS with adjusted WFC (0.7-1.0 ppm) and the rest having PWS with natural WFC which can be accurately measured. Future studies may better show whether an increased level of WFC offers effective protection against the development of AD in late life, as suggested by Still and Kelley.<sup>2</sup>

### CONCLUSIONS

The stupendous complexity of AD makes it unlikely that study of a single risk factor like aluminum neurotoxicity will lead to effective prevention and treatment of this lethal brain disease. Many other risk factors must also be considered. Among these are the aging process, amyloid deposition, blood-brain barrier defects, genetic predisposition, head injury with loss of consciousness, immunologic disorders, infectious agents (prions and slow viruses), endocrinopathy, toxic agents and vascular disorders. As yet, there is no proof of a causal association between aluminum

intake, aluminum neurotoxicity, and the presence of aluminum deposits in the AD brain. Nonetheless, further studies of fluoride intake offer a novel approach to the discovery of a potentially effective means of prevention of AD. □

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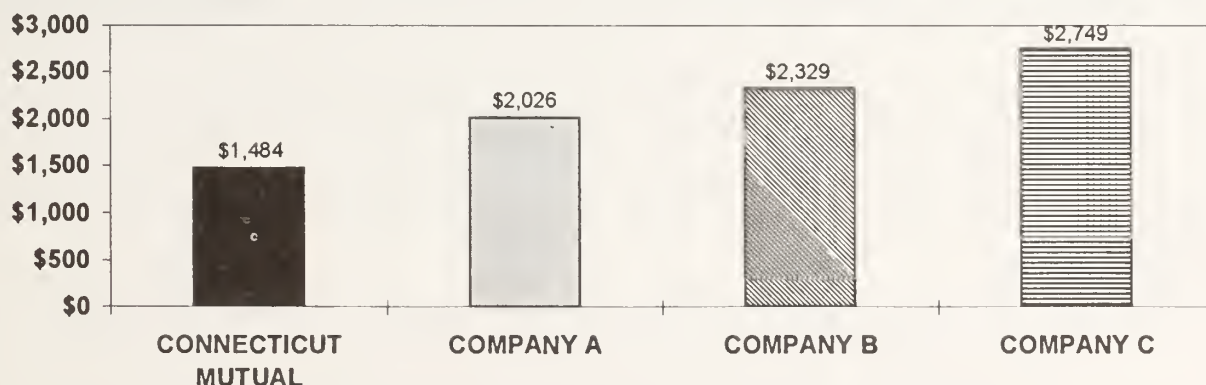
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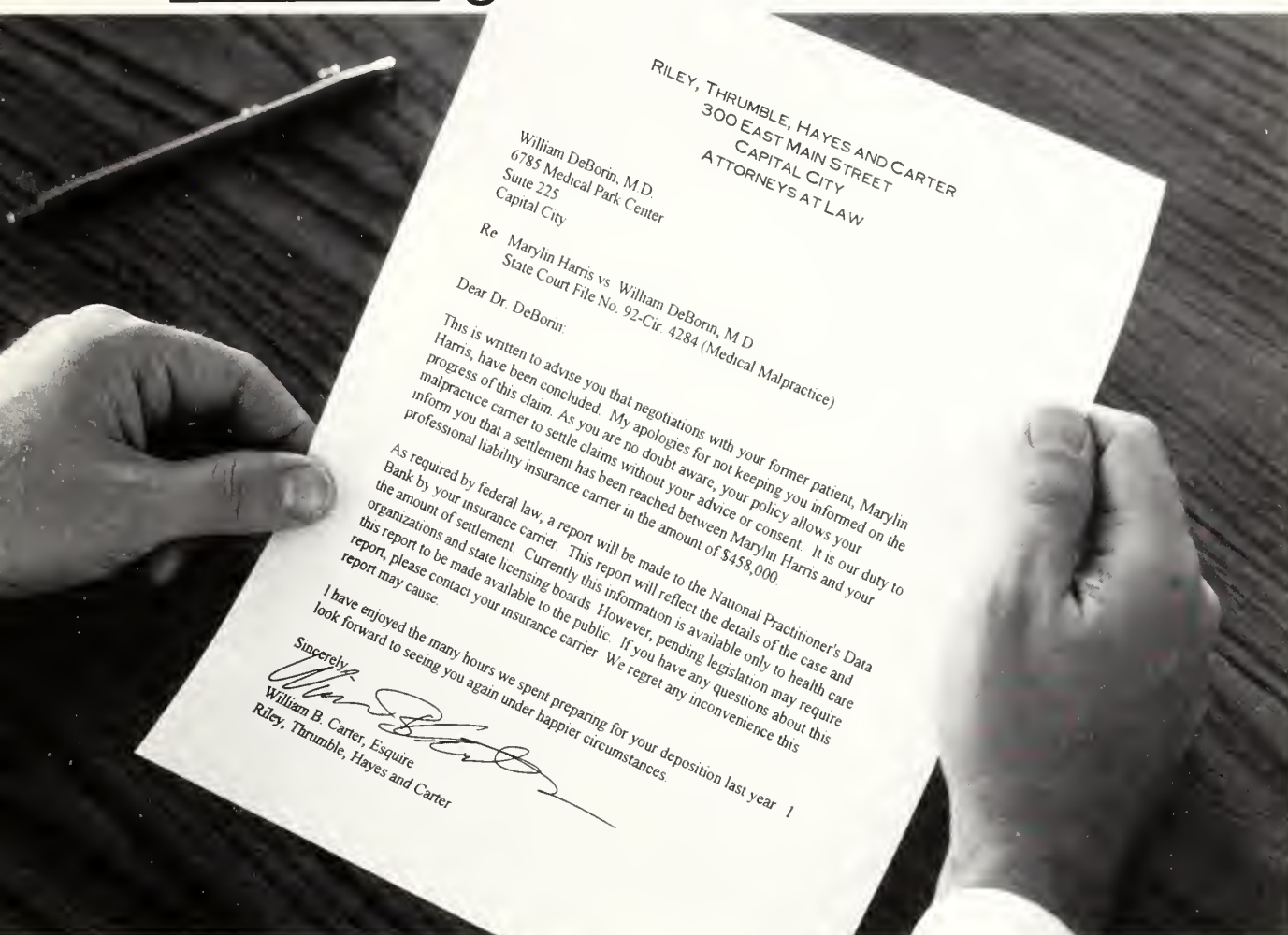
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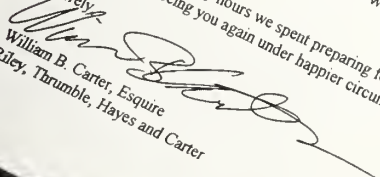
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I have enjoyed the many hours we spent preparing for your deposition last year. I look forward to seeing you again under happier circumstances.

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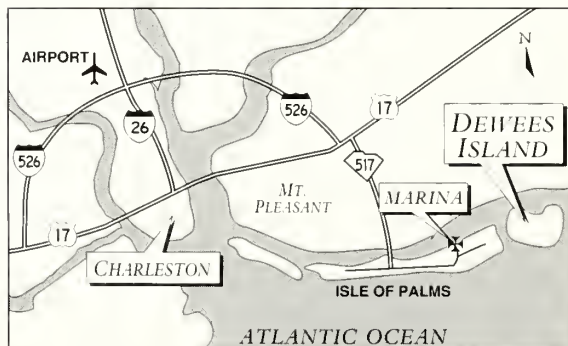
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Babcock founded the National Association for the Study of Pellagra, and served as president from 1909 to 1912. This association sponsored three international conferences on pellagra, all held in South Carolina. With Dr.

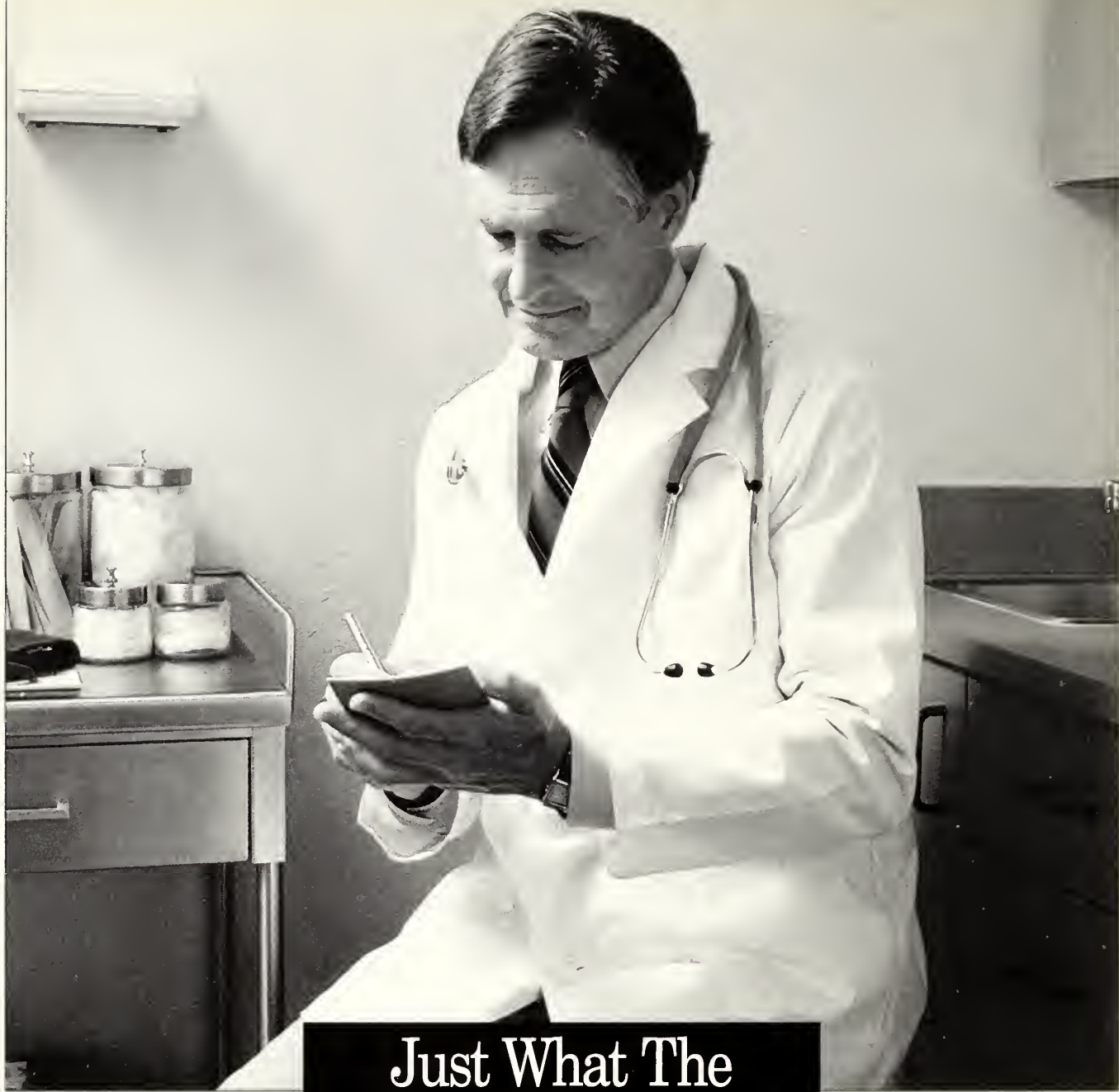
Lavinder of the Public Health Service, Babcock translated into English Dr. A. Marie's *La Pellagre*. Except for Joseph Goldberger, Babcock did more than anyone else in America to bring this disease to the attention of the profession and the public.

Babcock was born in Chester on August 11, 1856, the son of Dr. and Mrs. Sidney Babcock. He was educated at Exeter and Harvard; and after specializing in "alienism," he spent four years at the McLean Hospital in Waverly, Massachusetts. He returned to South Carolina in 1891 to become the superintendent of the Lunatic Asylum, a position he held for the next 23 years. His leadership made this institution one of the best in the country.

Dr. Babcock was a professor of psychiatry at the Medical College of the State of South Carolina. During his final years, he was the founder and administrator of the Waverly Sanitarium in Columbia.

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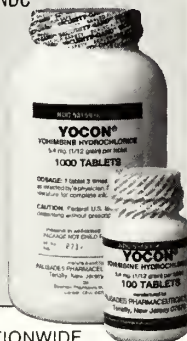
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## INDEX TO ADVERTISERS

Anne M. Bowen, C. F. P.....	572
The Cliff's at Glassy.....	Cover 3
Companion Technologies.....	529
CompuSystems, Inc. ....	Cover 4
Deweese Island .....	568
Lexus of Charleston .....	538
MedCorp Health Systems, Inc. ....	548
Medical Protective Company.....	567
Palisades Pharmaceuticals.....	572
Nationsbank .....	570
PHP Healthcare Corporation.....	530
Providers' Billing Services.....	Cover 2
Ultimate Auto Connection .....	547
U. S. Air Force Reserve .....	564
Winchester Surgical Supply .....	559

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(2) Paid or Requested Mail Subscriptions (Include Advertisers' Proof Copies Exchange Copies)	4763	5075
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  - Be sure to furnish all information called for in item 15 regarding circulation. Free circulation must be shown in items 15d, e, and f.
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VOLUME 90  
NUMBER 12  
DECEMBER 1994  
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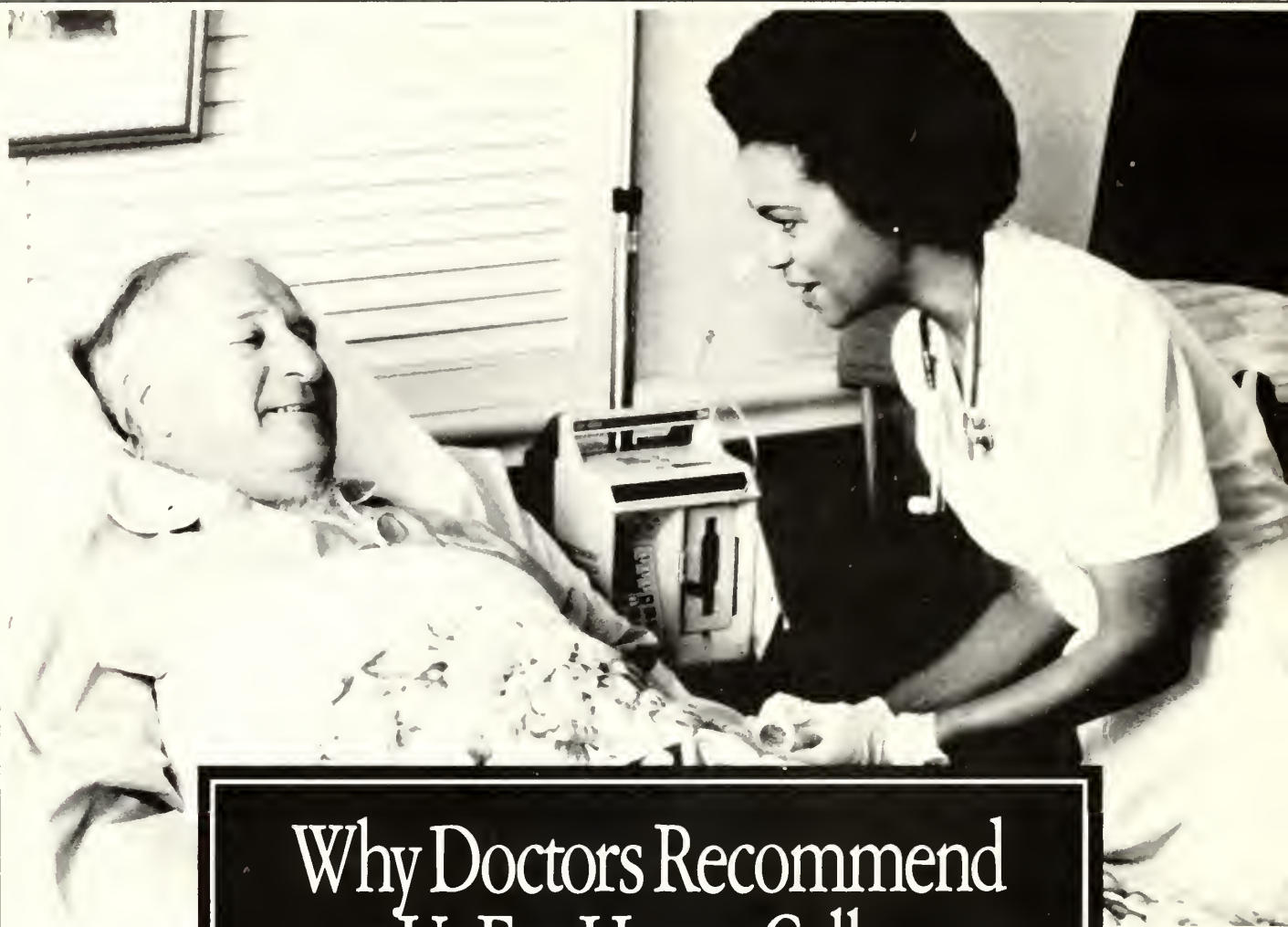
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# President's Page

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## THE PALMETTO HEALTH INITIATIVE

South Carolina, like many other states, is attempting to move Medicaid beneficiaries into managed care plans. Specifically, Governor Carroll A. Campbell, through the Health and Human Services Finance Commission (HHSFC), has submitted a section 1115 waiver request to the Health Care Financing Administration (HCFA) allowing South Carolina to expand Medicaid benefits to individuals at or below 100 percent of the federal poverty level. The Palmetto Health Initiative (PHI) is intended to slow the intolerable rise in cost of the state's Medicaid program, provide appropriate care for more uninsured, low income residents, and secure a medical home for all Medicaid beneficiaries.

Your SCMA staff and leadership have been involved in the waiver development since October, 1993. Governor Campbell and Eugene A. Laurent, Ph. D., Executive Director, HHSFC, deserve great credit for including physicians early in the planning process instead of at the moment of implementation which is estimated to be early 1996. States such as Tennessee are having turbulent beginnings for their Medicaid expansions because they did not get early and adequate provider input.

The South Carolina proposal initially allowed only one option for doctors and patients. This plan would have offered a fully-capitated product available for contract only to large managed care conglomerates or health maintenance organizations (HMOs). The SCMA insisted on having other payment methods included in the proposal and was successful in convincing the Governor's staff and HHSFC to include a primary care case management option. Such a program provides a capitated rate only for primary care services with all other specialists billing via the existing fee-for-service arrangement. This alternative allows the provider to deal directly with the HHSFC regarding payment and services rather than through a managed care company or HMO intermediary.

The impact of the PHI on our state's health care delivery system will be enormous. Over a half million of our citizens will precipitously be thrust into a full or partially capitated payment environment. At last count, over 20 new HMOs are considering PHI as an open door to an assured source of initial clients and enrollees.

SCMA staff is working closely with the HHSFC as it completes the milestones leading to full implementation, and will continue to monitor all pertinent developments. To help you keep abreast, the SCMA established the Physicians' Hotline To The Palmetto Health Initiative (1-800-825-7821) this month. Please call with your questions or comments about Medicaid managed care in South Carolina.

*O. Marion Burton MD*

O. Marion Burton, M. D.  
*President*

## Contents

### Original Scientific Articles

- 579 Kidney Function in Congestive Heart Failure**  
*Arthur V. Williams, M. D.*
- 586 Managed Care for the Frail Elderly: The Pace Project**  
*E. Paul Eleazer, M. D., Judith Pinner Baskins, R. N., John Egbert, M. D. Plt. D., C. David Johnson, M. D., Lisa Wilson, M. D.*
- 592 Renal Colic due to Henoch-Schönlein Purpura**  
*William Lane M. Robson, M. D., Alexander K. C. Leung, M. B., B. S., Michael S. Mathers, M. D.*

### Special Article

- 601 You can do Something About Adolescent Substance Use and Abuse: A Practitioner's Guide to Adolescent Alcohol and Other Drug Prevention**  
*N. Peter Johnson, Ph. D., Benjamin O. Stands, M. D., Harry S. Strothers, III, M. D.*

### Editorials

- 611 Third World Cardiology: Back to Basics**  
*N. B. Baroody, Jr., M. D.*
- 613 "The Rights of the Child" Revisited**  
*Charles S. Bryan, M. D.*

### Features

- 621 Alliance Page**
- 617 Letters to the Editor**
- 620 On the Cover**
- 577 President's Page**

### Association

- 607 CME Calendar**
- 615 Gray Matter**
- 622 Index to Volume 90**
- 596 Physician Recognition Award**
- 597 SCMA Newsletter**

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## KIDNEY FUNCTION IN CONGESTIVE HEART FAILURE

ARTHUR V. WILLIAMS, JR., M. D.\*

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Concepts of congestive heart failure have changed and broadened during the last several decades. Originally thought to be a redistribution of extracellular fluid (ECF) volume secondary to failure of the right or left ventricles it is now recognized that multiple endocrine and neurologic changes are activated by signals from the failing heart. Pivotal to reply from these signals, the kidney is then responsible for changes in systemic vascular resistance and control of ECF volume, both critical to cardiac preload and afterload. Therapeutic modulation of volume and resistance has improved the prognosis of congestive failure. This review gives a brief history of concepts of heart failure and discusses the role of the kidney in its pathophysiology and principles of therapy.

The state of the art of the physiology of congestive heart failure was summarized by Dr. Ernest H. Starling in a lecture delivered to the Royal College of Surgeons of England in 1897. He began by considering the mean systemic pressure, approximately 10 mm Hg, in arteries and veins in the absence of the heart beat. He then described the increase in pres-

sure in the arterial circuit secondary to peripheral vascular resistance and the lowered pressure in the venous system as the heart resumes beating. He attributed the large increase in pressure in the arterial circuit that might occur with the addition of a small amount of volume to that circuit's relatively small volume as compared with that of the venous side. As a corollary the venous circuit has small pressure changes with the addition of volume because of its relatively large capacity. Starling was aware of the rise in pressure in heart failure in large veins, but his experimental methodology, the measurement of limb volume, led him to believe that pressure in smaller veins and capillaries is normal in heart failure induced by the pericardial injection of oil. He concluded that the plethora of heart failure is not due to increased venous pressure and states, "We must, therefore, seek for some other explanation of dropsy in this disease." At no point was fluid retention secondary to alteration in renal function considered.<sup>1</sup>

By 1940 the understanding of the mechanics of heart failure had progressed. Isaac Starr and his associates developed a mechanical device simulating both right and left ventricles and pulmonary and systemic circulations. This device led them to question the then current

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mechanical displacement of blood into the pulmonary bed being the major event in congestive failure. They concluded that, "Mechanisms which cause an increase of static blood pressure, such as general vasoconstriction, pressure on vessels from without or the insertion of additional fluid within the vessels must be introduced before the pressure relations of clinical congestive heart failure can be reproduced."<sup>2</sup> By 1943 Dr. Starr had a model that he hoped would reproduce heart failure in a dog by destruction of the wall of the right ventricle by a cautery. Surprisingly, little or no increase in venous pressure followed. In his discussion he says that "as one abandons the conception of a direct mechanical relationship between heart disease and congestive failure and tries to substitute a chain of events one must concede that there may be causes of congestive failure other than heart disease....If the chain goes by way of the kidney renal lesions this might cause it."<sup>3</sup>

Further understanding of the role of the kidney in the genesis of heart failure was accelerated during World War II by the development of the flame photometer that facilitated the analysis of sodium in blood and urine. This was designed by W. R. Domingo, director of a soil laboratory in Kampen, Holland for the purpose of analyzing the salt content of land reclaimed from the Zuiderzee. Through his acquaintance with Willem J. Kolff this instrument was introduced into medicine with photometers built by Domingo for the Hammersmith Hospital in London, the Queen Wilhelmina Hospital in Amsterdam and other institutions.<sup>4</sup>

During the following two decades a formidable mass of information accumulated regarding circulatory physiology, salt and water balance and exchange of fluid and electrolytes through cell membranes. Otto H. Gauer and his associates summarized information available in 1970. Their discussion includes volume control by physical and autoregulatory mechanisms including stretch and osmo-receptors and their translation of these messages into a neuroendocrine mecha-

nism of regulation of extracellular fluid volume.<sup>5</sup> This summary contains the basic mechanisms for the current interpretation of congestive heart failure.

By 1980 mechanisms of body fluid homeostasis in congestive heart failure were well understood. Seminal research was contributed by Barry M. Brenner and his associates investigating proximal tubular sodium recovery as a result of changes in hydrostatic and oncotic pressures across the proximal tubule.<sup>6</sup> They documented the fact that the sequel to increased peritubular oncotic pressure was increased movement of salt and water from lumen to peritubular capillaries. Initiating this event was increased efferent arteriolar resistance that increased glomerular hydrostatic pressure and, therefore, increased glomerular filtration rate. The effluent blood from the glomerulus into the peritubular capillary was thus concentrated, its protein content increased, and its hydrostatic pressure decreased. Both of these changes result in avid proximal salt reabsorption. A summary of the effect of peritubular Starling forces, renal nerve activity in congestive failure and humoral effector mechanisms were compiled by Brenner and Skorecki in 1981.<sup>7</sup>

The kidney responds appropriately to a variety of stimuli to initiate the sequence of events described above, the afferent limb beginning in the atria, the arterial tree and the central nervous system. The response is as if there were a state of hypovolemia. The volume control system is said to respond to the relationship between cardiac output and peripheral vascular resistance. Arterial blood volume may be diminished relative to the volume of the arterial tree by a decrease in cardiac output in heart failure or diminished resistance as in hepatic disease.<sup>8</sup>

A major signal for the renal response to heart failure is decrease in the cardiac index. In a study by renal responses investigated in three groups of patients with cardiac indices of greater than 2L/min per square meter (group A), between 1.5 and 2 (group B), and less than 1.5 (group C). Glomerular filtration

rates were decreased in all three groups but preserved better than renal blood flow as would be anticipated with a relative increase in efferent resistance except in group C where the drop in GFR appeared to parallel to the fall in blood flow with the filtration fraction tending to fall. The reason for this was not clear. Renal vascular resistance was increasingly severe as the cardiac index fell with no disproportionate increase in efferent tone in the patients with the lowest cardiac indices. As evidence of increased sympathetic tone, all patients had elevated plasma levels of noradrenaline. Plasma renin activity and aldosterone were increased in all patients with no significant differences between the groups.<sup>9</sup>

Angiotensin II may initiate salt retention in heart failure by vasoconstriction of the efferent arteriole with the series of events described above leading to proximal recovery of sodium. It is known that this hormone is also a potent stimulator of aldosterone by the adrenal glands. The role of aldosterone in the pathogenesis of heart failure has been questioned. Aldosterone may not be elevated and its suppression by a converting enzyme inhibitor may not change sodium balance. However, the administration of spironolactone in patients with stable heart failure with normal glomerular filtration rates consistently results in a negative sodium balance in spite of slight increases in plasma aldosterone, renin activity and norepinephrine during its administration. Spironolactone is a specific aldosterone antagonist. These results would indicate that aldosterone is an important factor in sodium retention. The increase in proximal reabsorption with limited sodium delivered distally, the site of action of aldosterone, may account for failure of sodium balance to "escape" aldosterone effect in heart failure.<sup>10</sup>

Another mechanism for tubular sodium recovery is efferent renal nerve stimulation in congestive heart failure. Virtually all segments of the nephron receive noradrenergic innervation. Sodium and water retention occur via a direct response of the tubule and may occur with no demonstrable change in renal hemo-

dynamics.<sup>11</sup> Normally a left atrial receptor stimulates reflex efferent renal nerve activity that causes return to normal sodium balance after salt infusion into an animal. In rats with heart failure following coronary artery ligation this effect is blunted resulting in salt retention on low salt diets. This effect is abolished by renal denervation.<sup>12</sup>

In 1979 A. J. Debold described granularity in atrial cells that changed with water and electrolyte balance. These cells later proved to be the source of a potent natriuretic and diuretic substance, atrial natriuretic hormone (ANH).<sup>13</sup> In the kidney this factor acts on specific receptors in the renal microvasculature and proximal and distal tubular epithelium to induce hyperfiltration, inhibition of sodium transport and the suppression of renal release responsible for natriuresis, diuresis and diminished blood pressure. In addition, the effect of antidiuretic hormone (ADH) on water reabsorption is blunted.<sup>14</sup>

It is paradoxical that in heart failure when natriuretic factor is high in serum, urine salt concentration is low. Moreover, in this condition the hormone plasma level may respond normally to atrial stretch or increased central volume by increasing lower body pressure by an antigravity unit without concomitant saluresis. The cause of this attenuated response in heart failure is at least due in part to the presence of the multiple antinatriuretic and antidiuretic forces that are more important determinants of sodium excretion than plasma ANP concentration. This does not mean that elevated levels of ANP are not playing a role in body fluid in such patients and, eventually, therapeutic maneuvers maintaining high levels may prove to be beneficial.<sup>15</sup>

The action of arginine vasopressin's water retention in heart failure has been reviewed. Hyponatremia is the clinical hallmark of its nonosmotic release.<sup>16</sup> However, the less well known vasoconstrictor property of the hormone may represent the evolutionary basis for its release in response to circulatory stress.<sup>17</sup>

In addition to its action of conservation of body water by facilitating its reabsorption



from the collecting tubular lumen and vasoconstriction it also stimulates synthesis of prostaglandin E2. This eicosanoid decreases the flow of water through the tubular cells modifying vasopressin action. To further complicate the system of checks and balances that attempt to stabilize volume during congestive heart failure, aldosterone is known to abolish the prostaglandin E2 effect on water movement by inhibition of its synthesis.<sup>18</sup>

Prostaglandins have other actions including vasodilation, vasoconstriction, mesangial contraction or relaxation (changing glomerular capillary surface area available for filtration) and Na-K-ATPase inhibition or stimulation in addition to its effect on water metabolism noted above.<sup>19</sup>

Indomethacin does not decrease glomerular filtration rate in hypertensive patients or mildly volume expanded dogs, but previous volume restriction in proteinuric patients with normal GFRs results in decreased filtration. In congestive failure, where E2 is a potent renal vasodilator, the kidney behaves as it does with volume depletion and the administration of prostaglandin synthetase inhibitors may result in acute oliguric renal failure.<sup>20</sup> Renal effects of this group of drugs is uncommon but in heart failure they may also interfere with excretion of water, sodium and potassium and with diuretic and anti-hypertensive therapy. These risk factors for side reactions associated with NSAIDs have prompted the search for one that is kidney sparing. Although sulindac produces fewer complications in at-risk patients, renal side effects have been observed. Renal function should be checked in patients using NSAIDs with a short half life after a few days and after one to two weeks after using a drug with a long half life such as sulindac.<sup>21</sup>

The kinins are also active in regulation of renal blood flow, the most noted being bradykinin. These compounds increase renal blood flow, mediate the hyperfiltration of high protein diets, inhibit the response to ADH and net sodium reabsorption, and induce the release of renin in isolated glomeruli. It is of

clinical interest that converting enzyme inhibitors (CEI's) also inhibit kinases prolonging the life of bradykinin.<sup>22</sup> There is controversy as to whether the prolongation of the life of bradykinin by CEIs contributes a beneficial effect to patients with heart failure. However, since bradykinin is formed locally it may be impossible to quantify changes even if bradykinin's activity is enhanced.<sup>23</sup>

Dopamine is a precursor of norepinephrine and exerts pronounced effects of its own on the cardiovascular system and the renal vasculature. Its receptors cause renal vasodilation with increase in blood flow as well as natriuresis. In addition, cardiac afterload is reduced. The effect of dopamine on the kidney is both direct and indirect. Increased cardiac output and, secondarily, increased renal blood flow and GFR result in diuresis and salt loss. In addition specific receptors in renal tubular cells directly cause diuresis and natriuresis.<sup>24</sup>

Endothelins are the most recently described vasoconstrictors. These peptides are elevated in patients with heart failure. Plasma elevations are not correlated with elevation of atriopeptin, BUN or creatinine. Concentrations in the range of 5 to 40 pmol/ml are vasoactive and may be pathophysiologically significant in congestive heart failure.<sup>25</sup>

## **TREATMENT OF CONGESTIVE FAILURE BY MODIFYING RENAL MECHANISMS**

The relationship between the physical and humoral factors at play in heart failure is such that it is not possible to alter one without compensatory change in another designed to protect circulatory integrity. Diuretics decrease volume but increase angiotensin II concentration. Angiotensin II stimulates prostaglandin production and the use of converting enzyme inhibitors decreases prostaglandin production tending to cause renal vasoconstriction. All drugs used in therapy must be monitored because of this complex interplay.

Converting enzyme inhibitors, however, are now commonly used in the treatment of congestive failure. In 1987 the results of the coop-

erative North Scandinavian Enalapril Survival Study was published. The prognosis of congestive heart failure at the time was 50 percent annually. Two hundred fifty-three patients with severe congestive heart failure were assigned to a double blind study to receive either a placebo or enalapril. Those receiving the placebo were continued on their previous medication including the use of other vasodilators. The crude mortality at the end of 6 months was 26 percent in the enalapril group and 44 percent in the placebo, a reduction of 40 percent. By the end of the study there had been 68 deaths in the placebo group and 50 in the enalapril, a reduction of 27 percent. There was no difference in the incidence of sudden death between groups. There was a 50 percent reduction in death due to heart failure. The most common adverse effects were hypotension and increasing serum creatinine. Other factors that lead renal function to become dependent on angiotensin II are high dose diuretics, hyponatremia and preexisting renal impairment. Hyperkalemia was more common in the enalapril group but there was no relationship between this and renal failure or mortality. With this encouraging study a trial of angiotensin converting enzyme inhibitors became routine therapy in the management of congestive failure.<sup>26</sup>

The mechanisms of action of converting enzyme inhibitors are complex. They prolong the action of bradykinin but the significance of this in moderating renal blood flow and function is uncertain. Increase in bradykinin does seem responsible for the side effects of the drugs such as rash, cough and angioneurotic edema. Bradykinin is also responsible for prostaglandin release. This should be beneficial in congestive failure but compelling evidence is not present. Treatment with ACEIs can reverse left ventricular hypertrophy due to reduction in afterload but it also inhibits the growth of vascular smooth muscle cells induced by angiotensin 2, also reducing LVH. The proposed benefits of these drugs in heart failure by novel mechanisms require further investigation.<sup>27</sup>

Treatment of heart failure with non-specific vasodilators had been done prior to the enalapril study, notably, that of the Veterans Administration study reported in 1986 comparing prazosin, combination of hydralazine and isosorbide dinitrate and placebo, all combined with standard therapy. Prazosin alone was of no benefit. However, the combination therapy compared with prazosin and placebo had a favorable effect on left ventricular function and mortality.<sup>28</sup> A second Veterans Administration Study comparing enalapril with hydralazine-isorbide dinitrate was begun in 1986.<sup>29</sup>

These and other long-term studies were recently reviewed with the conclusion that nearly all patients with congestive heart failure and impaired left ventricular function should be treated with vasodilators and that "ACE inhibitors may be superior to other vasodilators in the treatment of congestive heart failure."<sup>30</sup> In patients with high plasma aldosterone levels six month's mortality is significantly higher than in those with below median levels. And, in spite of treatment with an ACE inhibitors mortality rates or plasma aldosterone levels may remain high. In addition to aldosterone's salt conserving effect there is evidence that the mineralocorticoid specific receptors in the myocardium binding aldosterone are responsible for myocardial fibrosis. Blocking this action with spironalactone prevents fibrosis in the hypertensive rat. Combined therapy with spironalactone and ACE inhibitors has been demonstrated to be useful and well tolerated so long as renal function is not impaired. Low doses of both drugs are used and serum potassium levels are closely monitored. However, prospective, controlled studies of ACE inhibitor therapy with and without spironalactone are needed.<sup>31</sup> It might be reasoned that since angiotensin II causes efferent arteriolar constriction and subsequent proximal tubular salt retention, that blocking angiotensin II production would allow more salt to reach the site of diuretic action distally and to amplify the effect. However, rapid compensation of volume depletion

by increased sympathetic activity and changes in renal blood flow, GFR and aldosterone stimulation quickly blunts diuretic effect. The intermittent use of diuretics with time for interim return of volume status may promote an unwanted diuresis. It has been suggested that in essential hypertension daily use of diuretics in combination with an ACE inhibitor can be "expected to result in less unnecessary urinary sodium loss than might result from intermittent or erratic use of these medicines."<sup>32</sup> Presumably this is true in heart failure although long prospective studies are needed.

Dopamine, a sympathomimetic amine, a precursor of norepinephrine, may increase renal excretion of salt and water. Extensive clinical research has been conducted to evaluate the usefulness of dopaminergic stimulation to improve the hemodynamic and neurohumoral abnormalities of heart failure. Several dopaminergic agonists such as ibopamine, dopexamine, levodopa and fenoldopam have been included in these studies. Stimulation of these receptors may inhibit release of renin, aldosterone and norepinephrine.<sup>33</sup> However, their use in clinical therapy of congestive heart failure has not been widely accepted.

## CONCLUSIONS

Multiple endocrine and neurologic changes are activated by signals from the failing heart. Central to response from these signals is the kidney that becomes responsible for change in ECF and peripheral resistance, both critical to the achievement of appropriate preload and afterload in congestive heart failure. Since manipulation of these factors have accounted for the recent improvement in the prognosis of congestive failure, the role of the kidney in congestive heart failure has been reviewed. □

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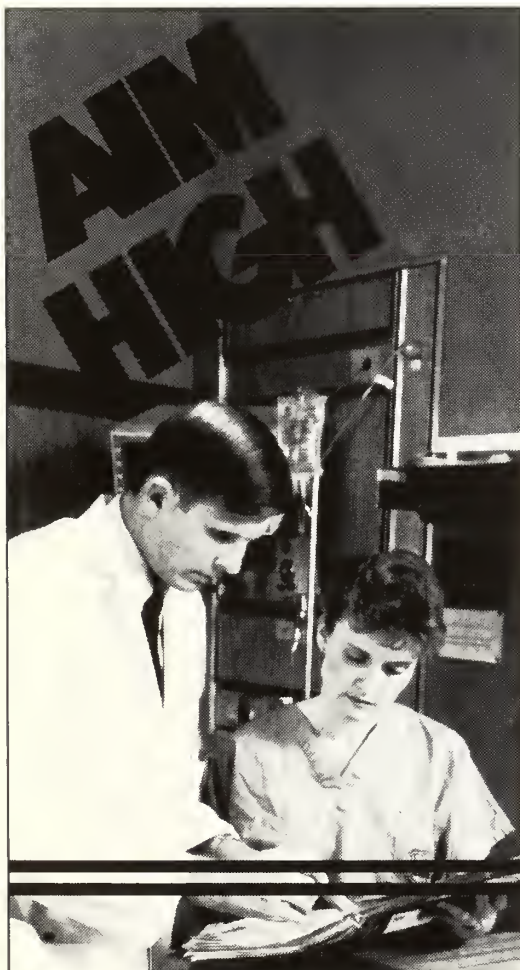
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# MANAGED CARE FOR THE FRAIL ELDERLY: THE PACE PROJECT

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The graying of the population is likely to have a major impact on health care in America. While Federal (Medicare) dollars fund much of acute care for older patients, state funding (Medicaid) provides many of the services for long-term care. The increase in the elderly population is likely to increase both of these costs significantly. For example, Medicare costs for the oldest old may increase six fold by the year 2040 (in constant 1987 dollars).<sup>1</sup> Cost-effective care for our aging population is particularly challenging for those frail elderly persons who meet nursing home level of care criteria. The PACE (Program For All-inclusive Care Of The Elderly) Project is a managed care system which integrates acute and long term care, in-patient and out-patient care, while also integrating Medicare and Medicaid financing to provide cost effective all inclusive care to elderly participants. This article will provide historical background on the PACE Project, describe the model of care implemented in PACE programs, and provide an update on Palmetto SeniorCare, one of the first four PACE sites.

## BACKGROUND

In the late 1960s, a group of concerned citizens from the Chinatown/North Beach/Polk Gulch district in San Francisco recognized the need for care for the frail elderly in their area. Initially this group was planning to develop a

nursing home to serve the area. In 1971 a consultant, Marie Louise Ansak, was hired to study the feasibility of building a nursing home in the district. However, Ms. Ansak instead recommended developing a community based system of out-patient services for the frail elderly (similar to that used in the English day hospital system). The first day health center opened in March 1973 and provided elderly participants with meals, personal care services, rehabilitative services, and recreational activities as well as nursing, social and transportation services. A multidisciplinary team approach of caring for the elderly was developed. The philosophy of the program was and is to maintain participants in the community for as long as medically, socially and economically feasible. The service package provided was modeled on institutional Long-Term Care, with the added social, behavioral, and morale benefits of continued life in the community. Primary medical care was still provided by the participant's local physician. Often, multiple physicians, health care institutions, and other care providers were involved in episodic care. Despite strong efforts to assure adequate communication and continuity, by 1978 it was apparent that this fragmented system of health care delivery was inefficient and often not in the participant's best interest. At that time, primary medical care was added to the model, including both in-patient and out-patient services. This integrated model proved successful in meeting the needs of the frail elderly population in a cost effective and efficient

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manner.<sup>2,3</sup> The success of On Lok attracted the attention of many of those seeking answers to the nation's health and long term care needs. However, it was felt that ethnic, cultural, and geographic factors unique to the On Lok program were so critical to On Lok's success that it was questionable whether this model could be replicated elsewhere.

In 1986, Congress approved legislation which would enable further study of the On Lok model in other geographic areas and in ethnically diverse populations. PACE (Program For All-inclusive Care of the Elderly) was developed to begin demonstration projects throughout the country based on the On Lok model. Initially five sites were approved (subsequently this was extended to 15). Initial replication sites included those in East Boston, MA; Portland, OR; Milwaukee, WI; Columbia, SC; and the Bronx, NY. Besides determining the feasibility of replicating the On Lok model of care in different geographic and ethnic regions, PACE was also to evaluate patient outcomes for enrollees and cost effectiveness of the care provided. Currently the national project has approximately 2,000 enrolled participants representing the geographic areas noted above. A more detailed description of the individual sites has been published by Dr. Robert Kane in his article on a qualitative analysis of the PACE program.<sup>5</sup>

Palmetto SeniorCare in Columbia was one of the initial sites selected to participate in PACE. Initially opening its first Day Health Center in the Eau Claire section of Columbia, Palmetto SeniorCare has now grown to 195 participants and has two additional centers. The program is sponsored by two agencies, Richland Memorial Hospital and the South Carolina Department of Health and Environmental Control. Funding to start up costs was provided through the Robert Wood Johnson Foundation. The site has been closely affiliated with the Division of Geriatrics at the University of South Carolina School of Medicine and serves as a training site for residents in Internal and Family Medicine, medical students, and students of multiple other disci-

plines.<sup>6</sup> In the startup phase, the program developed required components as a fee for service provider, billing under established guidelines for covered services. Subsequently it entered a three year demonstration period during which time it functioned under waivers\* with progressive risk sharing from the Health Care Financing Administration (HCFA) and the Health and Human Services Finance Commission (HHSFC). The program was successful in the demonstration phase and is now established under waivers\* from Medicare and Medicaid. For each participant, the program receives a capitated amount of money per month to provide all services to that participant. The program has received national recognition for its success in implementing the On Lok model of care.<sup>7</sup>

### THE MODEL

At a time when "managed care" has become a buzzword, the PACE/On Lok model with its focus on multidisciplinary assessment, coordination of services, and participant needs is one of the truly integrated modes of health care delivery in the country. The On/Lok PACE model represents a fundamental shift in the way that care is delivered, using a multidisciplinary team familiar with each case to target services rather than relying on reimbursement guidelines and consumer preferences to provide this targeting of resources. The overall result is the use of a broader range of services tailored and coordinated to support specific team and participant objectives.

Each participant in the PACE system receives an evaluation by a physician, nurse, social worker, nutritionist, activity/recreational therapist, and a rehabilitation specialist.

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\*Waivers: The term waiver was coined to describe a number of financing initiatives which have in common that a participant in the program forgoes or "waives" some portion of their normal benefits under an entitlement program in favor of the services provided under the waiver. In the PACE projects, the participant waives his/her right to seek covered services whenever and wherever he/she wishes in the fee-for-service system in favor of a more comprehensive service package provided through the PACE program.



Some sites also include routine assessments by pharmacy, optometry, audiology, dentistry and podiatry. Participants are evaluated at intake and then quarterly to determine their health care needs. In addition, unscheduled re-evaluations are performed whenever there is a major change in the participants status or plan of care.

After individual assessment by team members, the entire interdisciplinary team gathers to discuss and develop a care-plan for the participant. Participants and their caregivers are involved in all phases of this process. Unlike fee for service where rigid rules on eligibility and reimbursement have a major impact on any care planning process, the PACE program allows maximum flexibility for the team to negotiate details of the plan of care with participants and their families. This flexibility allows for negotiation of such things as in-home services (personal care aids, homemaker services, meals, etc.), additional day health attendance, and respite care. Decisions are based on the assessment of a participant's needs, in conjunction with the team's analysis of the resources necessary to meet those needs within available settings, including the participant and the participant's caregivers' ability to provide for themselves.<sup>2</sup>

Nowhere is the power and flexibility of the model more evident than in the management of acute illness. While hospitalization is frequently an important part of the management, skillful use of team resources, day health center services, home care, and the support of the participant's caregivers usually results in a care plan which reduces or eliminates hospital stays while simultaneously producing comparable or better functional outcomes. In addition, the reduction of institutional care reduces iatrogenic illness and limits the disruption of everyday life which can be as devastating as the illness itself for many geriatric patients. Since many families have repeatedly experienced hospitalizations of one to two weeks or longer as the first step in managing all serious acute illnesses, education of participants and caregivers and attention to their anxieties and

concerns are as important as the actual provision of services in implementing home based care. The coordination which characterizes PACE care is developed through constant attention to effective group process within the team. A more detailed description of the model is provided in C. Eng's article.<sup>2</sup>

The PACE model is a unique service and financing package. Table 1 summarizes some of the significant differences among fee-for-service care, traditional health maintenance organizations, and the PACE model. Capitated payment systems are a familiar form of reimbursement for HMOs and similar care systems which capitate physician payments with one monthly fee regardless of the intensity of service delivered to the patient. The PACE model is a capitated system where this concept is extended to the full service package. This provides a financial incentive for coordinated care and against over utilization. The team process itself, community involvement, participant satisfaction review, and internal and external review systems all provide incentives against under utilization.<sup>5,8</sup>

In most fee-for-service systems, there is limited long term care coverage. This lack of coverage is a problem and is one of the major issues confronting those proposing health care reform. HMOs also have limited or no coverage of long term care benefits. The PACE model includes long term care and combines it with acute care thus resulting in a truly integrated and "seamless" health care delivery system. The cost of nursing home placement (when required) is covered completely and primary care services and care management continue to be provided by the program.

The three systems also differ in whom they target for service. The fee for service system targets the general population. Health maintenance organizations generally target the healthy individual while the PACE project specifically targets the frail elderly. At the present time, it is unclear whether the PACE model will be applicable in less frail elderly or other populations (feasibility studies are exploring implementation of the model in frail

TABLE 1

	<u>Traditional Care</u>	<u>HMO</u>	<u>PACE</u>
Reimbursement	Fee-for-service	Capitated	Capitated
Long-term Care Coverage	Limited or none	Limited or none	All inclusive acute and LTC
Target Population	General population	The healthy	The frail elderly
Physician/Patient Ratio	1:2,000-5,000	1:1,000-5,000	1:75-90
Physician/Location	MD office	MD office	Day health center
After Hours	Emergency room	Emergency room	Home or emergency room
Preventive Services	Limited	Prevention emphasized in most	Heavy emphasis on prevention
Health Care Delivery System	Fragmented	More coordinated	Total coordination and managed care

children and AIDS patients and the disabled adult greater than age 55).

The location of service delivery is another major difference between the PACE model and fee for service or HMO. The PACE model emphasizes the day health center approach. Patients receive their routine care, episodic care, and other medical and rehabilitation services in the same location where they are coming for routine social day care services. After hours coverage may be provided in the emergency room but is frequently provided in the patient's home instead. Acute care service may be delivered in the hospital but many acute medical illnesses (not requiring intensive care unit service) are managed at home, in boarding homes (or other similar housing), in the day health center, or in a nursing home.

In the fee-for-service system, only a limited number of preventive services are reimbursable. The PACE model places a heavy emphasis on prevention including addressing specific geriatric syndromes. Primary, secondary, and tertiary prevention are all routine for PACE programs.

Fragmentation is a frequent problem in traditional health care delivery. Care is rendered in an uncoordinated fashion depending on payor source, location of service delivery, individual patient insurance coverage, and the knowledge of the patients, their informal caregivers, and of the health care providers involved in the patient's care. The PACE

model provides a coordinated delivery system in which all aspects of the patient's health care are managed by a team of individuals who know the patient's condition and the patient's and family's wishes in detail.

The interdisciplinary team is a major difference between PACE and more traditional health care systems. By virtue of frequent team meetings and extensive contacts among team members, the participants, and their caregivers, each discipline involved in the care of the participant has an unusually broad perspective. Thus, the physician practicing the PACE model has a much better understanding of the social situation and whether families or other caregivers will be able to provide a specific type of care for a given patient, while the social worker practicing in the PACE model has a more detailed picture of the participant's medical condition and prognosis, and how this is likely to stress the informal support network. For example, the decision whether to hospitalize or not to hospitalize a patient with pneumonia requires detailed knowledge of family dynamics, family resources, availability of home health, after hours and weekend coverage, and backup systems should the initial plans for care not fall in place. In the fee-for-service system, few if any physicians have time to handle this complex set of issues. However, in the PACE model an entire team works with the family and participant to effectively deliver coordinated care of high quality

in a very efficient manner. In addition, intervention with family and caregivers in such areas as home safety, neglect, nutrition, exercise and psychosocial functioning can lead to reductions in morbidity for the participant.

## RESULTS

The final evaluation for the PACE project will take several years. However, we have already learned several things from the demonstration portion of the project. First, the On Lok model of care can be effectively implemented in a variety of geographic and ethnically diverse areas. Second, the project has met with a high degree of acceptance by clients, caregivers, and providers both within PACE and in the broader community. An initial qualitative analysis of the PACE project indicated the early success of the replication sites.<sup>5</sup>

A recent study by the South Carolina State Health and Services Finance Commission found a high degree of satisfaction with the services offered by Palmetto SeniorCare. Seventy-two participants were initially selected at random. Forty participants and 30 caregivers were interviewed. Seventy-four percent of respondents indicated that they felt that their medical care was excellent to very good. Most of the respondents (63 percent) felt that the medical care that they were receiving under this project was an improvement over the care which they were previously receiving. Caregivers felt that the program had given them increased social time, improved their family time, and resulted in reduced amounts of stress. The "human" side of the program was evident in participants reporting that they had made new friends at the center (97.5 percent), enjoyed the food (87.5 percent), and enjoyed the activities (95 percent). Ninety percent reported that their quality of life had improved since enrolling!<sup>6</sup>

While cost effectiveness has yet to be completely evaluated due to limited data, comparisons to the current methodology used to determine the program's reimbursement at least form some basis for determining effectiveness. Currently Medicare reimbursement

is based on the Area Average Per Capita rate for the given area multiplied by a frailty factor. Each site nationally is reimbursed at 95 percent of this rate.

Preliminary cost comparisons have been favorable. In South Carolina, costs for Medicaid were tracked period for persons age 55 and older who reside in a nursing home facility in Richland County, (State and Federal nursing homes were excluded). The program then was reimbursed at 73 percent of that cost. Thus a minimum of a five percent savings to Medicare and 27 percent savings to Medicaid which is mandated by the lower reimbursement level. Monthly participant expenditures for Palmetto SeniorCare are approximately 68 percent of the cost for an equivalent Medicare, Medicaid (dually eligible) patient in the fee-for-service system.

The key to the cost effectiveness of the model, is the ability of the program to shift care from the more costly acute care setting to that of subacute or community based settings. The greatest challenge is to accomplish this without compromising the quality of care. The coordination and flexibility of PACE care planning can dramatically reduce the need for acute hospital services. Over a three-year period, Palmetto SeniorCare reduced average hospital utilization for enrolled participants by more than 80 percent, and average length of stay by 50 percent. The primary care physician can implement an earlier discharge without compromising quality of care because he/she knows the patient will be picked up by the program's van and brought into the Day Health Center, where they will be seen by the same physician. With the use of center staff and home care, medication compliance is greatly improved.

The need for other types of institutional care is also reduced by the improved coordination in care. All clients who enter a PACE project must meet the state definition of nursing home level of care. Yet, across the PACE project, the rate of nursing home placement is only 4.5 percent, a figure which includes temporary use for caregiver respite as well as long term



placements.

While cost effectiveness has yet to be completely evaluated due to limited data, comparisons to the current methodology used to determine the program's reimbursement at least form some basis for determining effectiveness. Currently, Medicare reimbursement is based on the AAPC rate for the given are multiplied by a frailty factor. Each site nationally is reimbursed at 95 percent of this rate for the Medicare portion.

While not the answer to all long-term care needs in the state, the model does offer alternatives and choices for the most number of families who do wish to care for their loved ones in their own home and community.

## CONCLUSION

The demographic imperative requires that we look for cost-effective means for caring for the elderly population. One alternative for caring for frail elderly patients is the PACE model. This model focuses on integration of

financing, integration of care, and prevention of disability with an improved quality of life. Other populations may benefit from the same approach. □

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# RENAL COLIC DUE TO HENOCCH-SCHÖNLEIN PURPURA\*

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## INTRODUCTION

Henoch-Schönlein purpura (HSP) is a vasculitic disease with an immunological pathogenesis that commonly involves the kidneys.<sup>1</sup> Non-renal involvement of the urinary tract has been reported but is considered to be uncommon. We report a six-year-old boy with HSP who developed renal colic associated with the passage of tubular-shaped blood clots in the urine.

## CASE REPORT

TB was born after an uncomplicated pregnancy, labor, and delivery. His birth weight was 3.38 kg. At the age of five weeks, he developed projective vomiting. Pyloric stenosis was diagnosed, and the child underwent a pyloromyotomy (Ramstedt's operation).

At the age of six years, he developed a nasal discharge, cough, and fever. Approximately one to two weeks later the patient experienced abdominal pain, vomiting, aching in the legs, and a rash over the feet. On admission to hospital, his weight was 20 kg (20th percentile), height 118.5 cm (35th percentile), blood pressure (BP) 98/50 mm Hg, heart rate 120 beats per minute, respiratory rate 28 per minute, and temperature 37.6° C. He had mild anterior cervical lymphadenopathy, a grade 1 to 2 functional ejection systolic murmur at the left

sternal border, tenderness and guarding to palpation of the lower abdomen, swelling of the ankles and feet, and a petechial and purpuric rash over the feet and the right knee. Blood tests on admission revealed a hemoglobin of 115 g/L, white blood cell (WBC) count of  $24.0 \times 10^9/L$  with 63 percent neutrophils, 18 percent bands, 16 percent lymphocytes, one percent monocytes, and three percent atypical lymphocytes, platelet count  $383 \times 10^9/L$ , sodium 128 mmol/L, potassium 3.8 mmol/L, chloride 96 mmol/L, total carbon dioxide 28 mmol/L, urea 6.5 mmol/L, creatinine 55  $\mu\text{mol/L}$ , total protein 31 g/L, albumin 14 g/L, glucose 7.1 mmol/L, calcium 1.86 mmol/L, and phosphorus 1.43 mmol/L. A throat swab culture was normal. The anti-streptolysin O titre (ASOT) was 166 Todd units. The prothrombin time and partial thromboplastin time were normal. The third and fourth components of the serum complement were 1.30 g/L and 0.30 g/L, respectively. The total hemolytic complement was 46,000 units per liter. The serum immunoglobulin G (IgG) was 5.96 g/L, immunoglobulin A (IgA) 2.62 g/L, and immunoglobulin M (IgM) 0.64 g/L. The urinalysis revealed a random specific gravity of 1.035, 3+ protein by dipstick, and greater than 100 red blood cells (RBC), 10 to 15 WBC, five to seven WBC casts, three to five granular casts, and eight to 10 hyaline casts per high power field. A diagnosis of HSP was established, and he was treated with prednisone 2 mg/kg/day for one month.

Sixteen days after admission, he developed severe abdominal pain. The abdominal pain was colicky in nature and localized to the left

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side of the abdomen. He was noted to writhe on the hospital bed and cry during these episodes. In between the colicky pain, the patient had a generalized, persistent, and dull-aching epigastric and periumbilical abdominal pain. In association with the episodes of colicky abdominal pain, the patient was noted to have gross hematuria with contained blood clots that were tubular-shaped. He continued to develop episodes of left-sided colicky abdominal pain for three days. A renal ultrasound revealed mild hydronephrosis on the left side. An intravenous pyelogram and a retrograde pyelogram were considered, but the condition of the patient did not permit these studies. A repeat renal ultrasound one month later continued to show mild left hydronephrosis, unchanged from the previous ultrasound.

During the hospital admission, he also developed protein-losing enteropathy requiring intravenous hyperalimentation, bruising of the left scrotum and tenderness of the left testicle, focal seizures with twitching of the left hand, hematochezia, anemia (6.6 g/L) requiring transfusion, hypertension requiring treatment with anti-hypertensive medications, nephrotic syndrome, and pulmonary edema. The patient developed renal insufficiency with the serum creatinine and urea increasing to a maximum of 156  $\mu\text{mol/L}$  and 24.1  $\text{mmol/L}$ , respectively. The patient was hospitalized for two months. On discharge, the blood creatinine and urea were 66  $\mu\text{mol/L}$  and 21  $\text{mmol/L}$ , respectively, and the total serum protein and albumin were 48 g/L and 29 g/L, respectively.

One year after the initial presentation, the BP was 110/60 mmHg, and the urinalysis revealed 2+ protein by dipstick and 10 to 20 BC and 2 to 5 WBC per high power field. The blood creatinine was 71  $\mu\text{mol/L}$ , urea 5.9  $\text{mmol/L}$ , creatinine clearance 131  $\text{ml/min/1.73m}^2$ , and 24-hour urine collection for protein 707 mg. Fifteen months after the initial presentation, he developed painless gross hematuria and generalized edema in association with pharyngitis. The patient did not

develop abdominal pain, joint pain or swelling, or a rash. The throat swab grew group A beta hemolytic streptococci, and the ASOT was 833 Todd units, falling to 625 Todd units two months later. The blood creatinine and urea were 78  $\mu\text{mol/L}$  and 19.8  $\text{mmol/L}$ , respectively. A 24-hour urine collection revealed a creatinine clearance of 98  $\text{ml/min/1.73m}^2$  and 3845 mg of protein. The serum total protein and albumin were 50  $\mu\text{mol/L}$  and 26  $\text{mmol/L}$ , respectively. He did not experience any further relapses, and, 11 years after the acute presentation, the creatinine clearance was 85  $\text{ml/min/1.73m}^2$ , the 24-hour urine protein was 438 mg, and an ultrasound and a Mag 3 nuclear renal scan were normal without any evidence of hydronephrosis.

## DISCUSSION

Kidney involvement is reported in up to 60 percent of patients with HSP.<sup>2</sup> Most patients with renal involvement present with microscopic hematuria. Painless gross hematuria is also common. Hematuria is usually due to an endocapillary proliferative glomerulonephritis. The patient we report presented with gross hematuria associated with renal colic, the passage of tubular-shaped blood clots in the urine, and mild hydronephrosis observed by renal ultrasound. The location and quality of the pain suggest an origin from either the kidney or the ureter. The tubular shape of the blood clots observed in the urine suggests that the blood clots were formed in a ureter. The possibilities to account for this presentation include significant renal bleeding due to glomerulonephritis or renal papillary necrosis, or ureteral bleeding due to ureteritis. A renal source of the bleeding with subsequent clot formation in the ureter is a possibility, but in our view, is unlikely. Gross hematuria due to glomerulonephritis is not associated with renal colic or clot formation. Renal papillary necrosis may be associated with renal colic due to the passage of a sloughed renal papillae, but no renal tissue was observed in the urine of our patient, which makes this possi-



bility less likely. We believe that the most likely explanation for the cause of the renal colic, gross hematuria, passage of tubular-shaped blood clots, and hydronephrosis in the patient we report is a ureteritis. HSP is a vasculitic disease with an immunopathogenesis that involves arterioles, venules, and capillaries.<sup>1</sup> In the patient that we report, it is possible that small vessels in the left ureter developed a leukocytoclastic vasculitis resulting in a ureteritis.

We reviewed the literature and identified 10 previously reported cases of ureteritis associated with HSP.<sup>3-10</sup> Ureteritis has been reported to develop in multiple segments of a ureter.<sup>3,6,9</sup> Previously reported patients with ureteritis have usually presented with gross hematuria and renal colic. However, Thompson et al. reported a six-and-a-half-year-old girl with HSP who presented with microscopic hematuria.<sup>4</sup> The authors did not mention the presence of renal colic, gross hematuria, or blood clots in the urine. An intravenous pyelogram (IVP) showed multiple filling defects in the right and the left renal pelvis and ureter.<sup>4</sup> The absence of colicky abdominal pain or gross hematuria in this patient raises the possibility that ureteritis may be more common than is generally appreciated in patients with HSP and that ureteritis may be responsible for microscopic hematuria in some patients. The demonstration of microscopic hematuria in patients with HSP is usually considered to be evidence of the presence of the endocapillary glomerulonephritis associated with HSP. We suggest that unless an examination of the urine sediment reveals the presence of cellular casts or dysmorphic red blood cells, ureteritis should be considered in the differential diagnosis of microscopic hematuria associated with HSP. The most common complication reported to be caused by the ureteritis associated with HSP is urinary obstruction.<sup>3,5-7</sup> Most of the previously reported cases of ureteritis presented with hydronephrosis and required surgical correction of the urinary obstruction.<sup>3,5</sup> Hydronephrosis requiring surgical exploration usually occurs during the acute ill-

ness but may develop up to four months after the onset of the HSP.<sup>3</sup> Urinary obstruction may be bilateral, and anuria due to bilateral urinary obstruction has been reported. Biopsies of ureteral tissue obtained at surgical exploration have revealed edema, mononuclear cell infiltrate, fibrinoid necrosis of the media and intima of the arterioles and capillaries, fibrosis, and calcification.<sup>3,5</sup> The hydronephrosis in our patient did not require surgical correction, and long-term follow-up has not revealed any evidence of persisting obstruction.

Prior treatment with a corticosteroid did not prevent the ureteritis in the patient that we report. Whether treatment with corticosteroid in our patient prevented the development of persistent obstruction requiring surgical correction cannot be determined. Several of the previously reported cases of ureteritis associated with HSP were treated with corticosteroid with varying results. Mougenot et al. reported a patient who appeared to respond to treatment with corticosteroid and who did not require operative intervention, although other authors have reported that treatment with corticosteroid did not prevent obstructive sequelae.<sup>3,6,8</sup>

Another complication reported due to ureteritis associated with HSP is perforation of the ureter. Grotte et al. reported a 10-year-old boy with HSP who developed ureteritis and in whom an IVP obtained one month after the onset of the HSP revealed dilatation of the right renal pelvis and extravasation of urine down along the right psoas muscle.<sup>5</sup> The patient that we report did not have any evidence of this complication.

A third complication reported due to ureteritis associated with HSP is calcification of retained blood clots in the pelvis of the kidney. Kher et al. reported a nine-year-old boy with HSP who developed ureteritis and in whom calcified blood clots were removed from the renal pelvis one year after the onset of HSP.<sup>3</sup> The patient that we report did not have any evidence of this complication.

Ureteritis is a potential complication of HSP and may be more common than is currently

appreciated. Patients with HSP who have microscopic hematuria but who do not have evidence of cellular casts or dysmorphic red blood cells should have diagnostic imaging studies performed to look for evidence of ureteritis. Ureteritis should be considered in patients with HSP who develop renal colic or who have tubular-shaped blood clots in the urine. An ultrasound of the urinary tract should be obtained in all patients with HSP who have microscopic hematuria in order to look for evidence of hydronephrosis, which may be a manifestation of ureteritis. Urinary obstruction requiring surgical correction is a possible sequelae of ureteritis associated with HSP.

### SUMMARY

A six-year-old boy with Henoch-Schönlein purpura (HSP) presented with renal colic and the passage of tubular-shaped blood clots in the urine. The authors suggest that the renal colic was due to a ureteritis. Ureteritis in patients with HSP may be more common than is generally appreciated. □

### ACKNOWLEDGEMENT

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## PHYSICIAN RECOGNITION AWARDS

The following SCMA physicians are recent recipients of the AMA's Physician Recognition Award. This award is official documentation of Continuing Medical Education hours earned.

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# SCMA NEWSLETTER

A PUBLICATION OF THE SOUTH CAROLINA MEDICAL ASSOCIATION  
Joy Drennen, Editor  
798-6207, in Columbia

Contributions welcomed  
1-800-327-1021, outside Columbia

December 1994

## HIGHLIGHTS OF THE NOVEMBER BOARD OF TRUSTEES MEETINGS

The SCMA Board of Trustees received a report concerning a meeting between SCMA board members and members of the SC Hospital Association regarding the formation of a Health Care Policy Committee. The board approved the formation of this committee whose purpose is to explore areas of common interest between SCMA and SCHA and work together on these common goals.

The board also announced a joint effort between SCMA and MUSC's Department of Health Administration and Policy. In early spring of 1995, SCMA's first Managed

Care Institute will take place in Charleston. The goal of the institute is to provide physicians with an extensive educational program about managed care. Watch this newsletter for further details.

The board also gave final approval to two ethical statements developed by SCMA's Medical Ethics Committee and a joint SCMA/SCHA Committee concerning ineffective or futile medical treatment and do-not-resuscitate orders. Watch for publication of these statements in a special issue of *The Journal* early next year. □

## MEDICARE UPDATE

Please read very carefully the December, 1994 *Medicare Advisory* which you should have received by this time. You should also have received the 1995 Fee Schedule and Participation Enrollment Package. You have the option until December 31, 1994 of changing your participation status for 1995. If you have questions, contact the Part B Provider Service Center at (803) 788-5568.

**Name Change:** Effective January 1, 1995, the South Carolina Medicare Divisions (Part A and B) will be known as **Palmetto Government Benefit Administrators, or Palmetto GBA**. Palmetto GBA is the official name for all government programs administered by Blue Cross and Blue Shield of South Carolina. When addressing mail to the Part B Division, you should use "Palmetto GBA/Medicare Part B." The mailing address has not changed.

**Provider Service Center Reorganization:** The Medicare Part B Provider Service Center has grouped related specialties together and created three teams, effective December 1, 1994. A complete breakdown by specialties is in the December, 1994 *Medicare Advisory*. Since teams are based on specialties, there will no longer be separate telephone numbers for participating and non-participating

providers. All providers should use (803) 788-5568 when calling the Provider Service Center.

**1995 Part B Deductible:** The 1995 Part B Deductible will not change but will remain at \$100.

**Basic Billing Workshop:** The next Medicare Part B Basic Billing Workshop will be held January 18, 1995 at the Embassy Suites Hotel in Columbia. To pre-register, see page 8 of the December, 1995 *Medicare Advisory*. The registration fee is \$30 per person.

**Informal Review by Telephone:** Did you know that you can request an informal review of your Medicare Part B claims over the telephone? The telephone appeals line is available Monday through Friday, 9:00 am to 12:00 noon, and 1:00 to 3:00 pm by calling (803) 788-0222, ext. 43839. Telephone appeals can replace written appeal requests on most types of claims involving ambulance services, radiology and pathology services, diagnosis code denials, and other similar claims denied with action code GT. Questions involving claims processing errors, billing questions, claim status inquiries, or corrected claims should continue to be directed to the Medicare Part B Provider Service Center. □

**Reminders for Filing Hard Copy Claims:** We strongly encourage providers to leave the upper right hand margin of the HCFA 1500 Claim Form blank. The Finance Commission microfilms each hard copy claim prior to being keyed and processed. When the claim is microfilmed, a claim control number is assigned and printed on the upper right hand corner of every HCFA Claim Form. If a claim form is submitted with anything printed in this corner, it is necessary to manually place labels over the information so that a claim control number can be assigned and printed. This manual step delays the processing and increases administrative costs.

Claim information must be present in the required fields before the data entry operators can properly key a claim.

## UPCOMING MEETINGS/WORKSHOPS

The SCMA has scheduled two workshops for physicians and their staff:

- 1. CPT Coding for Doctors' Offices, January 11, 1995, 8:30 a.m.-4:30 p.m.
- 2. Advanced Coding and Reimbursement Issues, January 12, 1995, 8:30 a.m.-4:45 p.m.

Presented by AMA Financing & Practice Services, Inc., both workshops will be held at the Sheraton Hotel and Conference Center in Columbia.

*For further information, please call Ginny Comer at the SCMA in Columbia at 798-6207 or toll free at 1-800-327-1021, ext. 242.*

The South Carolina Psychiatric Association has scheduled its 1995 annual meeting in the Ambassador Room of the Embassy Suites on Interstate I-26 at the Greystone Boulevard Exit in Columbia on Saturday, January 28, 1995. Registration fee is \$100 for psychiatrists, \$50 for residents and \$150 for all others. *For further information, call Cathy Boland at the SCMA in Columbia at 798-6207, or toll free at 1-800-327-1021, ext. 232.*

The 1995 SCMA Annual Meeting and Scientific Assembly will be held in Charleston at the Omni Hotel, April 20-23, 1995. Watch for a preliminary schedule of activities and registration information in February of next year. Meantime, mark your calendar now! □

If the information does not appear in the proper field, is unaligned, or too light to read, the claim will be returned with a cover letter.

**Use of 208 Forms:** The Finance Commission has received an increasing number of SHHSFC 208 Coinurance and Deductible Claim Forms with private insurance explanation of benefits (EOBs) attached. The 208 Form is **only** to be used with Medicare/Medicaid crossovers. It must be submitted with the **Medicare** EOB. A HCFA 1500 Claim Form with the insurance EOB attached is used to file for patients with private insurance and Medicaid. Offices may receive a letter regarding this issue if it is determined to be a continuing problem. □



## DOCTOR OF THE DAY

Volunteers are being signed up for "Doctor of the Day" for the 1995 Legislative Session.

If you can volunteer a day of your time any Tuesday, Wednesday or Thursday beginning in February of 1995, please call Jan McKellar or Barbara Garvin at SCMA Headquarters. Parking is provided and you will be introduced in both the House and the Senate.

This program is a service of the SCMA to the State House, and your participation is encouraged. Donations of medicines are also welcomed. □

## CAPSULES

The South Carolina Society of Ophthalmology was recently designated by the American Academy of Ophthalmology as a "1994 Model State Society," being only one of 10 state societies recognized to excel in organizational, political and public service projects during the past year.



ACMG, Inc. (Administrators for Physicians Care Network) will soon be conducting the annual fee analysis for comparison to the established PCN maximums. In early January, 1995, each individual physician will receive a copy of his/her fees currently on file and instructions pertaining to this process. Do **not** send your fees now. In the meantime, feel free to contact Client Relations at 771-0777 in Columbia or toll free 1-800-340-7494 should you have further questions.

PCN has been successful in being chosen as one of the networks to bid on major groups. PCN asks you to provide names of additional business contacts in your community and/or opportunities to present at local business/civic meetings. *Please call Barbara Whittaker or George O'Laughlin at SCMA Headquarters with these contacts.*

Four more hospitals have contracted with PCN: The Regional Medical Center of Orangeburg/Calhoun Counties, East Cooper Community Hospital in Mt. Pleasant, Spartanburg Regional Medical Center, and B. J. Workman in Woodruff, bringing the total number of hospitals to 33. Approximately 2,700 physicians have enrolled in the network to date. □

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### **PHYSICIANS' HOTLINE TO THE PALMETTO HEALTH INITIATIVE**

The SCMA has established a toll-free hotline,

**1-800-825-7821**

for physicians to call with their questions and comments regarding the Palmetto Health Initiative, a managed care program for Medicaid beneficiaries.

SCMA staff will respond to your questions or comments in writing within five working days. If you need an immediate answer, please indicate this in your message.

Selected questions and answers will be published in the SCMA Newsletter each month. The SCMA will also forward a copy of all questions and comments to Health & Human Services Finance Commission and Dr. Morrison Farish, the physician representative on the Legislative Medicaid Waiver Task Force. □

### **SCIMER AID TO BULGARIA**

The South Carolina Institute for Medical Education and Research (SCIMER) has collected and shipped a 40-foot container of used medical supplies and equipment to a hospital in Dobrich, Bulgaria.

Any physician wishing to donate used equipment or excess medical supplies should contact Melton Stuckey, MD, at (803) 254-2222. Dr. Stuckey will arrange for shipment to Bulgaria. The value of all items donated is tax deductible. □



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**PATIENT-PHYSICIAN RELATIONSHIP**  
**(From the DHEC Bureau of Drug Control)**

**Can an after hours, on-call physician establish a valid patient-physician relationship, prior to the issuance of a prescription, with a patient he or she has not seen?** While the establishment of a patient-physician relationship is not addressed, by statute or regulation, with respect to non-controlled substance prescriptions, Sections 508.2 and 514.2 of the SC Controlled Substances Regulation require that prior to the issuance of a prescription for a controlled substance in Schedules II, III, IV or V, "...the prescribing practitioner (physician) shall have a valid practitioner-patient relationship established with the recipient of the prescription...." These sections require that a physician have sufficient knowledge as to the medical need of the patient for such controlled substance, as well as make a good faith determination as to the identity of the patient.

After hours requests can often present a dilemma for an on-call physician, who may be confronted with an individual who indicates that he or she is a patient of the physician for whom the on-call physician is covering, or is a patient of another physician in the on-call physician's practice group. The individual will often indicate a medical condition for which a controlled substance would be appropriate therapy, possibly indicating that the unavailable physician has prescribed a particular controlled substance for this condition on one or more previous occasions. The on-call physician must then decide whether or not to comply with the request of the individual who may, in fact, be a drug seeker, or possibly refuse a patient who may have a legitimate medical need for a particular controlled substance.

**What then should a physician do in these situations in order to determine a legitimate medical need and make a good faith determination as to the identity of the patient, and thus establish a valid patient-physician relationship?** First of all, the physician should be wary of requests for a specific controlled substance, especially a request by brand name. While a patient may have been prescribed a particular controlled substance for an ailment in the past, most patients simply want relief from their discomfort, and will usually not make a specific request. On the other hand, drug seekers will usually make a specific request, and indicate that other similar drugs, usually in a lower schedule, either have not provided relief, or that they are allergic to them.

Secondly, the physician should be aware that drug seekers can often "recite the Merck manual" on a particular condition for which they are requesting a controlled substance, giving detailed descriptions of their symptoms. Thus, an individual who is "too knowledgeable" may, in fact, be a drug seeker, and not a legitimate patient.

Thirdly, the physician should ask the individual such questions as: (1) How long have you been a patient of Dr. X? (2) When did you last see Dr. X? (3) What is your chart number at Dr. X's office? (4) What is your address? (5) What is your phone number, so that I may call you after I call the pharmacy? (Calls from a pay phone should arouse suspicion.)

Fourthly, if the physician is still uncertain as to the legitimate medical need and/or the true identity of the individual, he or she should ask the pharmacist, at the designated pharmacy, about the patient profile of the individual, e.g., has there been any indication of drug seeking behavior before? Is this individual a regular patient of the pharmacy? While certainly not always true, most legitimate patients will utilize one pharmacy for all their prescription needs.

If the physician makes the decision to prescribe a controlled substance, the quantity should be limited to a few dosage units, since this is an emergency type situation, and the patient should be able to see the unavailable physician within 72 hours. It is further suggested that the physician ask the pharmacist to request identification from the individual picking up the prescription(s). Also, if the physician decides that the patient needs a non-controlled substance, in addition to the controlled substance, the physician should request that the pharmacist fill both prescriptions, or none at all.

Following these suggestions should assist the after hours, on-call physician in providing appropriate treatment for legitimate patients, and, at the same time, not be duped by a drug seeking individual. □

# YOU CAN DO SOMETHING ABOUT ADOLESCENT SUBSTANCE USE & ABUSE: A PRACTITIONER'S GUIDE TO ADOLESCENT ALCOHOL AND OTHER DRUG PREVENTION\*

N. PETER JOHNSON, PH. D.\*\*

BENJAMIN O. STANDS, M. D.

HARRY S. STROTHERS, III, M. D.

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## ALCOHOL AND OTHER DRUG RISK FACTORS

An understanding of risk factors is one means of determining the degree of danger to children. A number of factors are important in assessing the likelihood of children and adolescents becoming alcohol and other drug users. Presence of these factors does not mean that the patient will become a drug-user, but should alert you to the possibility. The absence of these does not assure that the youth will not use drugs.

### FAMILY RISK FACTORS

—*Alcoholism in parents or other siblings:* children whose parents or siblings are alcoholics/drug abusers are at double the risk of becoming substance abusers than those without that history.

—*Genetic factors:* children born to an alcoholic parent, even when raised by nonalcoholic foster parents, have much higher rates of alcoholism than those of nonalcoholic origins.

—*Cultural factors:* members of any group in which heavy drinking is socially acceptable, or strongly rejected – may also change drink-

ing patterns.

—Children from a family with a history of criminality or antisocial behavior are more likely to abuse drugs than those without such a family history.

—*Inconsistent parental direction or discipline:* unclear and/or inconsistent parental rules and reactions to their children's behavior, unusual permissiveness, lax supervision or excessively severe discipline, constant criticism, and an absence of parental praise or approval, are associated with higher rates of drug abuse in children.

—Divorce which results in *inconsistent parenting* by divorced partners or alcohol and other drug permissiveness by one or both parents.

—*Parental drug use or parental attitudes approving of drug use.*

—*Parent smoking, drinking heavily or abuse of other drugs.*

—Emphasis by practitioners on the *serious medical and social consequences of drug use* is usually more acceptable than discussions of legality.

### PEER RISK FACTORS

—Children whose *friends or siblings smoke, drink or use other drugs.*

—*Initiation into drug activities is usually through friends.* The local drug "pusher" is more likely to be a child's acquaintances who want to share the drug experience or "deal" to support their own drug use than a mysterious stranger lurking near the school.

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\*From the Department of Neuropsychiatry and Behavioral Science (Dr. Johnson), the Department of Family and Preventive Medicine (Dr. Johnson and Dr. Strothers), and the Department of Pediatrics (Dr. Stands, emeritus), The University of South Carolina School of Medicine, Columbia.

\*\*Address correspondence to Dr. Johnson at the Office of Alcohol and Drug Studies, Medical Library Building, Suite 310, University of South Carolina School of Medicine, Columbia, SC 29208.

### **ACHIEVEMENT, SOCIAL AND DEVELOPMENTAL RISK FACTORS**

—Children *unsuccessful in school* are more likely to become substance abusers than those who succeed. Children who are poor academic achievers are more likely to experiment with drugs earlier and become regular smokers, drinkers and abusers of other drugs.

—Adolescents *bored by school and disinterested in academic achievement* are more likely to be drug involved than those academically involved. Use of cocaine, other stimulants or hallucinogens is less common among teenagers with college plans.

—*Children who feel "at odds,"* strongly rebellious against adult authority and alienated from the dominant social values of their community are more likely to abuse drugs than those with strong bonds to family, church, synagogue and other conventional institutions.

—*Early antisocial behavior,* lack of social responsibility, fighting and other aggressive behaviors are predictive of later alcohol/drug abuse.

—*The earlier a child begins to smoke, drink or use other drugs,* the greater the likelihood of serious drug abuse later. Use of marijuana before the age of 15 greatly increases the probability of becoming a heavy user.

### **SCHOOL AND COMMUNITY RISK FACTORS**

—*Easy availability* of inhalants, tobacco, alcohol and other drugs.

—*Transitions* between schools

—*Labelling* and identifying students as "High Risk."

—*Economic and social deprivation.*

—*Low neighborhood attachment* and community disorganization.

—*Community laws and norms favorable to use* of alcohol and other drugs.

### **FACTORS WHICH HELP AVOID ALCOHOL AND OTHER DRUGS**

The task of the physician, parent, and the community is to reduce risk factors and enhance protective factors, the most powerful

of which is the family (see below). Positive social development:

### **FAMILY PROTECTIVE FACTORS (Stern, 1991; Family is the most powerful force.)**

—Mother sought *prenatal care* when pregnant.

—Close *bonding* with children.

—High *value placed on education or vocational training.*

—Parenting style is *high warmth/low criticism* rather than authoritarian.

—Parent is *nurturing and protective*.

—Parent practices *mood-maintenance skills,* keeping feelings in the mid-range rather than the extremes.

—Supportive relationships with *caring adults beyond the family.*

—*Shared family responsibilities.*

### **PEER PROTECTIVE FACTORS**

—Involved in *alcohol and drug-free activities.*

—*Respect* for authority.

—*Bonded* to conventional groups (eg., Boy Scouts, Girls Scouts).

—*Appreciation for unique talents* of each member of the group.

### **SCHOOL AND COMMUNITY PROTECTIVE FACTORS**

—*High expectations* for academics.

—School encourages *goal-setting and mastery* in all students.

—Staff views itself as *nurturing caretakers.*

—School and community encourages *altruism and cooperation.*

—School (and/or social organizations such as Scouts) *provides leadership and decision-making experiences.*

—School fosters *active involvement* of students.

—*Parents are involved* in the school through activities and process.

—Alternative alcohol and drug-free activities provided.

—Norms and *public policies support non-use*



by youth.

—Community provides *access to housing, healthcare, child care, employment, and recreation*.

—Community provides supportive networks and social bonds.

—Community involves youths in community service.

### PROGRESSION OF PROBLEMS

There is usually a progression of drug use, beginning with inhalants, alcohol and tobacco use, which usually precedes use of marijuana and other drugs. It is unusual for a child to experiment with sedatives, stimulants or hallucinogens without first having used alcohol, cigarettes or inhalants. Inhalants, alcohol, and tobacco, are viewed by experts as having a pivotal importance as “gateway drugs.” Youngsters who do not smoke or drink are 10 times less likely to use marijuana than those who do. Tobacco smoking thus becomes a major diagnostic clue for use of alcohol and other drugs. Some data indicate a three-fold difference in alcohol prevalence between youth aged 12-17 who smoke (60 percent) and those who do not smoke (20 percent).

### BEHAVIORAL SIGNS OF ALCOHOL AND OTHER DRUG USE

There are many nonspecific behavioral indicators which should alert you to the possibility that a young patient is using/abusing drugs. These add weight to the necessity for further interview and/or laboratory testing.

—*Sudden decline in school achievement.*

—*Change in extracurricular activities that were important to the child (athletics, Scouts, band, etc.)*

—*“Cutting” classes, tardiness or truancy from school.*

—*Marked personality changes.* Childhood and adolescence are marked by mood swings and some instability. Evidence of social withdrawal, marked changes in openness to communication with other family members, unexplained depression, changes in sleep patterns, sleeplessness, or excessive sleeping, are all

potential indicators of drug use.

—*Changes in personal appearance or hygiene.*

—*Marked changes in the peer group, especially if hanging around with known users or dealers.*

—*Increased secretiveness, unexplained phone calls, increased hostility to inquiry by parents, teachers, or physicians.*

—*Heightened social involvement* – “out every night.” Kids “hanging around” are more likely to be involved in drug use.

—*Unexplained disappearance of family funds, family or personal possessions may be related to obtaining money for drug purchases.*

—*Unexplained income and purchases of items for which parents can determine no legitimate funding source.*

—*Aggressive behavior* such as recurrent fighting, irritability, marked hostility, school delinquency and other evidence of social alienation.

—*Overt signs of alcohol and other drug use and/or intoxication.*

—*Sudden onset of use of over-the-counter preparations to reduce eye reddening may be a sign of marijuana intoxication or “snorting” cocaine.*

### ALCOHOL

At every stage of development, the most widely used drug is alcohol. Alcohol use has the highest public and mental health impact of any of the drugs used in America. Because of preoccupation with other drugs of abuse, alcohol abuse by teenagers is frequently overlooked, is minimized, or seen as part of simply growing up.

In reality, heavy drinking and alcoholism have been and continue to be our most serious adolescent and adult drug abuse problem.

Beer is the alcoholic beverage most commonly consumed by adolescents. Other alcoholic beverages popular with younger drinkers include citrus flavored “wine coolers” and mixtures of lemonade and alcoholic beverages. Part of their appeal is that the fruit flavoring masks the taste of alcohol and makes

them resemble carbonated soft drinks.

As with other drugs, the age of first use of alcohol has fallen regularly. During the 1930s the median age for drinking was between 17 and 19. Current surveys estimate that the *median age for first drink is age 12*. By the fourth grade, one quarter of children indicate that they feel pressure to drink. The National Household Survey in 1985 found that one in 10 children 12 to 13 years old had consumed alcohol during the month preceding the survey. Although drinking is illegal in all states for most seniors, seven of 10 are current drinkers; five percent consume alcohol on a daily or near daily basis. Nearly half of male high school seniors can be described as "problem drinkers" (having been drunk six or more times in the past year, or they acknowledge problems in *three out of the following five areas because of drinking: trouble with the principal, teachers or the police; difficulty with friends; driving after heavy drinking; or being criticized by a date for alcohol use*).

Twenty five percent of seventh graders report getting drunk at least once per year.

Tolerance to alcohol develops quickly — even after a few days of drinking — but is lost rapidly as well. Tolerance is the adaptation of the body and mind to the intake of a drug. Behavioral tolerance is the mental compensation made by an individual after the newness of the drug intake has passed. Adolescents have little to no experience. The occasional adolescent drinker can overdose on alcohol trying to keep pace with heavier drinking friends.

Since younger drinkers are also likely to be inexperienced, they are less capable of monitoring their alcohol use and less likely to be aware of their degree of impairment. As lighter weight persons than older drinkers, they are more likely to become drunk at lower levels of consumption.

Alcohol is the major cause of fatal and non-fatal traffic accidents. Teens and young adults (16-25) who represent only 22 percent of the driving population, are responsible for 44 percent of the fatal nighttime alcohol-related crashes. Size, inexperience and rapid alcohol

intake without accompanying food are likely to result in drunkenness that can be toxic, occasionally fatal.

Adolescents often *use alcohol with other drugs* which are metabolized along the same chemical pathways. As a result the combined effects are likely to be more prolonged and more toxic than alcohol alone.

Alcohol may cause a range of activities from fatal depression of the respiratory centers to more subtle impairment of psychomotor and cognitive functioning. Since alcohol readily reduces anxiety, and growing up is characterized by the need to learn to deal with many anxiety producing situations, the use of alcohol may become a chronic way of reducing anxiety and avoiding the demands of growing up.

*Early alcohol use* has been identified by researchers as a consistent predictor of later, more serious involvement with alcohol and other drugs. Thus, preventing early use may reduce the likelihood of both alcohol and other drug abuse as well.

## INHALANTS

Inhalants include products which can be sniffed or inhaled and are easily found at home or in grocery and hardware stores. Three types of inhalants are used: *aromatic hydrocarbons/solvents* (butane, gasoline, glue, old formula Whiteout, spray paint); *propellants* (nitrous oxide in whipping cream); *inert gases* (freon). Amyl and butyl nitrite ("Rush," "Hardware") have been legally sold until recently. One of the major effects is anoxia which causes a slight euphoriant effect. Some solvents are highly toxic to liver function, others can cause lethal suffocation. Rapidly expanding gases can absorb heat from body tissues and damage them. Other materials coat the lungs and can prevent oxygen exchange, or cause severe pneumonia. *Identifying the specific inhalant is important.*

## GENERAL

For adolescents, to screen for substance abuse consider using the R.A.F.F.T. designed by Pro-

### R.A.F.F.T QUESTIONS FOR ADOLESCENTS (Lewis, 1991)

- |          |  |
|----------|--|
| <b>R</b> | Do you drink or use drugs to <b>Relax</b> , feel better about yourself, or to fit in?  |
| <b>A</b> | Do you ever drink alcohol or use drugs while you are by yourself, <b>Alone</b> ?   |
| <b>F</b> | Do you or any of your closest <b>Friends</b> drink or use drugs?   |
| <b>F</b> | Does a close <b>Family</b> member have a problem with alcohol or other drug use?   |
| <b>T</b> | Have you ever been in <b>Trouble</b> from drinking or drug use (i.e., skipping school, bad grades, trouble with the law or parents)? |

ject ADEPT at Brown University (1991).

Common changes which practitioners and parents may observe related to alcohol and other drugs include:

- Decreased school performance and activities
- Changes in *behavior*.
- Changes in *types of friends* and associates.
- Changes in *language, dress*, and decorum.
- Decreases courtesy.
- Changes in *musical preference*.

Nurses in the physician's office can often spot these difficulties and inform the physician of potential difficulties.

#### PHYSICIAN AND PARENTAL TASK LIST FOR PREVENTION

- Raise the child with a strong self-image.
- Role model the behaviors and attitudes you wish the child to have – deal with parental alcohol problems immediately, for genetic and for behavioral reasons.
- Provide adult support and supervision.
- Provide anti-alcohol, tobacco, and other drug messages.
- Ask age-appropriate alcohol and other drug questions – the AAFP recommends starting with seven-year-olds, including pre-school exams.
- Be aware of community alcohol and other drug resources, and active in their development where needed.
- Identify high risk children early, and guide children from alcoholic homes to participate

in Alateen to prevent the ACOA syndrome from manifesting itself.

—Make aggressive diagnoses for adolescent substance use and problems.

#### HOW TO RESPOND

*Minimal Use:* If use has been isolated, the child needs evidence of reassuring adult love, rather than moralistic condemnation. The potential health hazards should be stressed in a fashion which does not condone use.

*Intoxication:* Make the point that intoxication at any age is undesirable, and especially while developmental processes are going on. The *potential negative impact on development of adult skills should be emphasized*.

The following may be useful for your work:

1. Johnson NP, Stands BO, Strothers HS, III. You can do something about adolescent substance use and abuse. Fighting Back informational pamphlet for physicians, Columbia, SC, 1993.
2. Johnson NP, Stands, BO. Physician referral of adult or adolescent patients with release of information without violating Part 2 CFR 42 confidentiality laws. Fighting Back informational pamphlet for physicians, Columbia, SC, 1993.
3. Johnson NP, Stands BO, Hines CJ, Black RM. Hints for identification of alcohol and other drug problems: A physician's guide (information booklet – community service organization). Columbia, SC: Fighting Back, 19 pp., 1992. □



# Millions Victimized by Family Members Every Year!

*Are you concerned about the effects of family violence and victimization within your community?*

*Become an advocate within your community for the prevention of family violence.*

Violence among family members has reached staggering proportions. Every year more than 2 million cases of child abuse and neglect are reported, between 2 and 4 million women are battered by their spouses, and between 700,000 and 1.1 million of the elderly population are abused.

The American Medical Association has formed a *National Coalition of Physicians Against Family Violence*. Through the *Coalition* the American Medical Association hopes to involve you in activities that address issues of child abuse, sexual assault, domestic violence and elder abuse because you have the unique ability to identify the symptoms, first-hand. By joining the *National Coalition* you will be showing your concern about the effects of family violence and victimization, and will become a committed advocate within your community for the prevention of family violence.

Through the *Coalition* you will:

- be informed about local contacts and referrals
- become aware of local and regional resources
- be provided with information regarding model educational programs
- become aware of treatment guidelines and protocols.
- have access to newsletters, public education materials and other publications
- receive an official membership card and frameable poster alerting your patients of your interest in and concern for this problem.

The only **cost** to you **is your commitment** to help curb this problem. Simply complete the membership application form below and mail to the Department of Mental Health, American Medical Association, 515 N. State Street, Chicago, IL 60610.

-----  
**Yes,** include my name in the *Coalition's* membership

Name \_\_\_\_\_

Address \_\_\_\_\_

City/State/Zip \_\_\_\_\_ Telephone # \_\_\_\_\_

Specialty \_\_\_\_\_

Auxiliary Member ☐ Yes ☐ No Other \_\_\_\_\_

Area of interest within Family Violence: ☐ Child Abuse ☐ Sexual Assault ☐ Domestic Violence  
☐ Elder Abuse ☐ Other

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# Continuing Medical Education

**First Quarter  
1995  
Calendar**

**James L. Haynes, M. D., Chairman**

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**Published by the SCMA Committee on Continuing Medical Education  
Post Office Box 11188, Columbia, SC 29211**

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Note: CME activities in neighboring states are listed when space permits.

## **JANUARY**

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**Thursday-Sunday January 26-29, 1995**  
**Charleston, SC, The Omni Hotel**  
***STFM Predoctoral Education Conference***  
**SPONSOR: STFM**  
**DESCRIPTION:** To teach family medicine faculty various clinical and educational aspects of instructing and coordinating predoctoral medical students.  
**CONTACT:** Priscilla Noland, (816) 333-9700, ext. 4510, FAX (816) 333-3884  
**CME CREDITS:** 14.25 AAFP Prescribed Hours

**Saturday January 28, 1995**  
**Columbia, SC, Embassy Suites Hotel**  
***1995 Annual Meeting of the SC Psychiatric Association***  
**SPONSOR:** William S. Hall Psychiatric Institute  
**CONTACT:** Cathy Boland, (803) 798-6207, ext. 232  
**PROGRAM FEE:** \$100 for psychiatrists, \$50 for residents, and \$150 for all others  
**FACULTY:** Drs. Eric Lister and Bert Pepper of APA's Office of Consultation Services  
**CME CREDITS:** 7 Hours, AMA Category 1

## **FEBRUARY**

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**Saturday February 4, 1995**  
**Charleston, SC, Basic Science Auditorium, MUSC**  
***1995 Anesthesia Update***  
**SPONSOR:** MUSC  
**DESCRIPTION:** Update in anesthesia.  
**TYPE OF AUDIENCE:** Anesthesiologists  
**CONTACT:** Barbara Baylor, (803) 792-1607  
**PROGRAM FEE:** TBA

**FACULTY:** Guest faculty and MUSC faculty  
**CME CREDITS:** TBA

**Monday-Saturday February 20-25, 1995**  
**Augusta, GA**  
***30th Annual Primary Care and Family Practice Symposium***  
**SPONSOR:** Medical College of Georgia  
**CONTACT:** Katrinka Akeson, (800) 221-6437 or (706) 721-3967  
**CME CREDITS:** 52 Hours, AMA Category 1

**Thursday-Saturday February 23-25, 1995**  
**Sea Island, GA**  
***Diabetes Update***  
**SPONSOR:** Medical College of Georgia  
**CONTACT:** Katrinka Akeson, (800) 221-6437 or (706) 721-3967  
**CME CREDITS:** 14 Hours, AMA Category 1

**Saturday February 25, 1995**  
**Greenwood, SC, Recovery Center**  
***5th Annual Update on Addiction Medicine***  
**SPONSOR:** Recovery Center and Self Memorial Hospital  
**DESCRIPTION:** Addiction medicine update to meet the need of primary care physicians dealing with addictive patients. Emphasis on occupational medicine issues.  
**TYPE OF AUDIENCE:** Family practice, general internal medicine, addictive medicine professionals  
**CONTACT:** Randy Cain, M. D. or Ann Griffin, (803) 227-4850  
**PROGRAM FEE:** \$50  
**FACULTY:** Laura Griffin, M. S., M. Ed., College of Charleston; Allen "Skip" Jones, Ph. D., University

of Mississippi; William "Win" Green, M. D., Greenwood, SC; Al Mooney, M. D., Willingway Hospital, Statesboro, GA  
CME CREDITS: 5 Hours, AMA Category 1

## MARCH

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**Friday-Saturday**                      **March 3-4, 1995**  
**Augusta, GA**

***Flexible Fiberoptic Sigmoidoscopy***

SPONSOR: Medical College of Georgia

CONTACT: Katrinka Akesson, (800) 221-6437 or  
(706) 721-3967

CME CREDITS: 14 Hours, AMA Category 1

**Monday-Wednesday**                      **March 5-8, 1995**  
**Charleston, SC, Mills House**

***Neonatal Nutrition***

SPONSOR: MUSC

DESCRIPTION: Update primary care physicians on latest advances in neonatal nutrition.

TYPE OF AUDIENCE: Pediatricians and family physicians

CONTACT: Diane Anderson, R. D., (803) 792-2112

PROGRAM FEE: \$130

FACULTY: Guest faculty and MUSC faculty

CME CREDIT: 17 Hours, AMA Category 1

**Thursday-Friday**                      **March 9-10, 1995**  
**Augusta, GA**

***Pediatric Advanced Life Support***

SPONSOR: Medical College of Georgia

CONTACT: Katrinka Akesson, (800) 221-6437 or  
(706) 721-3967

CME CREDITS: 14 Hours, AMA Category 1

**Monday-Tuesday**                      **March 13-14, 1995**  
**Charleston, SC, Hawthorn Suites Hotel**

***Radiology for Primary Care Physicians***

SPONSOR: MUSC

DESCRIPTION: Update primary care physicians on latest advances in radiology.

TYPE OF AUDIENCE: Family physicians and internists

CONTACT: Clydie de Brux, (803) 792-4267

PROGRAM FEE: TBA

FACULTY: Guest faculty and MUSC faculty  
CME CREDITS: 12 Hours, AMA Category 1

**Thursday-Friday**                      **March 16-17, 1995**  
**Augusta, GA**

***Advanced Trauma Life Support***

SPONSOR: Medical College of Georgia

CONTACT: Katrinka Akesson, (800) 221-6437 or  
(706) 721-3967

CME CREDITS: 17 Hours, AMA Category 1

**Sunday-Wednesday**                      **March 19-22, 1995**  
**Charleston, SC, Omni Hotel**

***Azaleas, Dogwoods and Racing Hormones***

SPONSOR: MUSC

DESCRIPTION: New aspects of the diagnosis and treatment of hormonally mediated disorders will be presented. The focus of the symposium will be to improve your understanding of these disorders and develop rational treatment strategies for their management.

TYPE OF AUDIENCE: Practicing gynecologists and obstetricians

CONTACT: Odessa Ussery, M. Ed., (803) 792-4071

PROGRAM FEE: \$350 before February 20; \$400 after February 20

FACULTY: Guest faculty and MUSC faculty

CME CREDIT: 13.5 Hours, AMA Category 1

**Monday-Tuesday**                      **March 20-21, 1995**  
**Charleston, SC, Sheraton Inn**

***HIV/AIDS: Education, Prevention and Management, 1995 Health Care Professional's Conference***

DESCRIPTION: National and regional experts will address current practical information that can be used in education and prevention strategies and in clinical management.

TYPE OF AUDIENCE: All health professionals

CONTACT: Odessa Ussery, M. Ed., (803) 792-4071

PROGRAM FEE: TBA

FACULTY: Guest faculty and MUSC faculty

CME CREDIT: 10.5 Hours, AMA Category 1

**Monday-Wednesday**                      **March 20-22, 1995**  
**Columbia, SC, Adams Mark Hotel**

***Hyperbaric Medicine 1995 Advance Training Seminar***

SPONSOR: USC School of Medicine



**DESCRIPTION:** Three day didactic program on thermal burns, brown recluse bites, hyperbaric oxygen and radiotherapy, skin flaps/grafts, hyperbaric oxygen in surgical infections.

**TYPE OF AUDIENCE:** Consulting hyperbaric physicians

**CONTACT:** Dick Clarke, (803) 434-7101

**PROGRAM FEE:** \$495 and \$395

**FACULTY:** State and out of state faculty

**CME CREDITS:** 18.5 Hours, AMA Category 1

**Wednesday-Sunday**                      **March 22–26, 1995**  
**Augusta, GA**

***Organ Procurement***

**SPONSOR:** Medical College of Georgia

**CONTACT:** Katrinka Akeson, (800) 221-6437 or  
(706) 721-3967

**CME CREDITS:** 14.5 Hours, AMA Category 1

**Friday-Saturday**                      **March 24–25, 1995**  
**Augusta, GA**

***Ophthalmology Resident - Alumni***

**SPONSOR:** Medical College of Georgia

**CONTACT:** Katrinka Akeson, (800) 221-6437 or  
(706) 721-3967

**CME CREDITS:** 10 Hours, AMA Category 1

**Sunday-Tuesday**                      **March 26-28, 1995**  
**Charlotte, SC, Mills House Hotel**

***5th Charleston Pulmonary Symposium***

**SPONSOR:** MUSC

**DESCRIPTION:** Update the clinician in pulmonary and critical care medicine. The course will emphasize the clinical approach to patients with pulmonary diseases utilizing pathophysiologic basis for rational therapy.

**TYPE OF AUDIENCE:** Pulmonologists and internists

**CONTACT:** Odessa Ussery, M. Ed., (803) 792-4071

**PROGRAM FEE:** \$325

**FACULTY:** Guest faculty and MUSC faculty

**CME CREDITS:** 11 Hours, AMA Category 1

**Wednesday-Friday**                      **March 29–31, 1995**  
**Augusta, GA**

***Functional Endoscopic Sinus Surgery***

**SPONSOR:** Medical College of Georgia

**CONTACT:** Katrinka Akeson, (800) 221-6437 or  
(706) 721-3967

**CME CREDITS:** 25 Hours, AMA Category 1

**Thursday-Friday**                      **March 30–31, 1995**  
**Charleston, SC, Hawthorn Suites Hotel**

***Digestive Disease Conference***

**SPONSOR:** MUSC

**DESCRIPTION:** Update on the management and treatment of digestive diseases.

**TYPE OF AUDIENCE:** Gastroenterologists, internists and family physicians

**CONTACT:** Odessa Ussery, M. Ed., (803) 792-4071

**PROGRAM FEE:** TBA

**FACULTY:** Guest faculty and MUSC faculty

**CME CREDITS:** TBA

**Thursday-Friday**                      **March 30-31, 1995**  
**Columbia, SC, Sheraton Hotel**

***12th Annual SC Conference of STDs***

**SPONSOR:** USC School of Medicine

**DESCRIPTION:** An update on the major SC STDs and will focus on interventions that are essential for the control of these diseases.

**TYPE OF AUDIENCE:** Physicians, registered nurses and social workers

**CONTACT:** Libby Green, (803) 737-4110

**PROGRAM FEE:** \$60

**FACULTY:** TBA

**CME CREDITS:** 8.5 Hours, AMA Category 1

## **CONTINUING MEDICAL EDUCATION COMMITTEE**

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171 Ashley Ave., Charleston 29425

Spence Taylor, M. D., Department of Surgical Education, Greenville Hospital System,  
701 Grove Road, Greenville 29605

**SPEAKER**—Roger Gaddy, M. D., P. O. Box 29, Winnsboro 29488

# Editorials

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*The following editorial by Dr. Baroody was based on his one-month tour of duty in a small hospital in the rural section of the Delta Region of Egypt. Guest editorials reflect the opinions of the authors and do not necessarily reflect the opinions of the officers and trustees of the South Carolina Medical Association.*

—CSB

## THIRD WORLD CARDIOLOGY: BACK TO THE BASICS

Four weeks of intense patient care in a third world country is a sobering experience. Intoxicated by the diagnostic plethora of supertech cardiology, there evolves a stuporous dependency on catheters and monitors, images and angiograms, pressures and pacers. We think with our calculators and computers and decide according to GUSTO, TIMI, CAST, SAVE or CONSENSUS. We rely on meta-mega studies to dictate to us specific and individualized and personalized therapy for our patients, many of whom we have cared about and cared for for 20 or more years.

When suddenly thrust into an archaic environment where basics is all there is, we begin to recruit and to reclaim age-old, time-honored talents in physical diagnosis—namely sight, sound, touch, and last but not least, history. Once again we engage in eye-to-eye contact with the patient, all the while observing skin color, neck veins, pulsations, and respiratory rhythms. Additionally, we feel carefully for the PMI and percuss the left heart and right heart borders for enlargement, feeling for thrills or precordial lifts. In short, we establish a relationship with the patient who happens to have a disease process rather than vicariously attacking a disease process that happens to reside in the patient.

In this medically primitive environment, we encounter many old acquaintances of years gone by. We listen carefully as whispered pectoriloquy quietly suggest to our faded memory the possibility of pulmonary consolidation. Our numbed fingertips are reminded that to

feel for the tip of enlarged spleen, we must press deeply in the left upper quadrant of the abdomen as the patient takes a deep breath. We are surprised to learn that patients with toes or fingers amputated may have not been victims of diabetes or peripheral ischemia but rather of leprosy. We become accustomed to feeling enlarged livers since most of the population in the Delta region of Egypt have schistosomiasis and secondary portal cirrhosis with portal hypertension. When they are admitted to the hospital with hematemesis, we anticipate bleeding esophageal varices rather than bleeding peptic ulcer disease as it is more commonly seen in the West.

To our surprise hypertension and arteriosclerotic disease are epidemic, with elevated cholesterols and heavy cigarette smoking as the rule rather than the exception. We have access to many of the effective cardiac drugs as well as antibiotics, so that appropriate therapy in these disorders is usually no problem. Lifestyle changes, however, seem imbedded deeply in the concrete of fixed cultural, economic and religious circumstances.

Suddenly, however, our therapeutic balloon explodes when we are confronted with unstable angina requiring immediate cardiac catheterization or tight mitral stenosis in need of cardiac surgery—both unacceptable options in light of the economic and social restrictions. The patient with chronic atrial fibrillation is deprived of anticoagulant therapy since INR levels cannot be monitored. Thrombolytic therapy is withheld from the patient with



acute myocardial infarction due to economic constraints, and once again, we regress into the therapy of the sixty's relying solely on bed rest and medications.

To be thrust into this arena of medical compromise and to have made a full-circle in 20th-century medical care is an experience not easily dealt with. To tell a mother that her nine-month old cyanotic child is in need of a \$25,000 corrective surgical procedure in another country is a cruel absurdity infiltrated with absolute truth. The hopelessness of such a scenario is abysmally factual, and we are left with a blue baby who is not yet dead.

Superstition has always been an archival adversary of medicine which in one instance presented in the grisly form of a mother pouring a glass of water down the throat of her young daughter, stuporous but recovering from a bout of hypertensive encephalopathy. In this act she literally drowned her own child because she did not want her to die in a state of thirst.

And so it continues in the third world, appropriately treating congestive heart failure with ACE inhibitors, digoxin and furosemide, while in the other room the macabre scenario of a mother inadvertently drowning her comatose daughter with a glass of water poured down her throat. Those in undeveloped countries need us. They need us to teach them, show them, instruct them, advise them and assist them.

They need to be told what is available to them and from this menu select what is practically appropriate for their existing circumstances.

We in turn need them to remind us of our aesculapian roots—where we came from, what we have forgotten that still works, and why we are bound together in this mutual conflict of man against disease.

N. B. Barody, Jr., M. D.  
McLeod Regional Hospital  
Family Medicine Center  
555 East Cheves St.  
Florence, SC 29506-2617

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## “THE RIGHTS OF THE CHILD” REVISITED

Why bother with organized medicine? Why go to meetings of the county medical society? Aren't there better ways to spend time?

Recently, I pondered William Osler's answers to these questions while driving to the scientific meeting of my county medical society. Grabbing his hat and coat one evening, Osler asked a younger colleague, “Aren't you going to the medical society?” “No,” the young man replied, “I think it's a waste of my time.” “Do you think I don't!” exclaimed Osler, and off he went. Osler knew that it's important to support one's medical society if for no other reasons than to promote good will and to participate in the issues-of-the-day. And he also knew that one might actually learn something!

Still, I doubted that I would learn anything of a useful nature that evening. The announced topic was: “Violence in Our Society: Prevention and Intervention.” True, the subject is extremely important and affects us all. But as physicians—individually or collectively—can we really make a difference? The speaker recited familiar facts. Each year, the 200 million guns in the United States account for the deaths of some 25,000 people, of whom 85 percent knew their killers in one way or another. Battery is now the leading cause of injury to women in the United States, and more than one in three women are abused during their pregnancies. Nearly two million elderly persons are abused each year. Well, what did I expect? After all, this was the medical society—not the Optimist Club!

Then the speaker took a different tack. The basic problem, he opined, is that more and more children are being raised in homes without love. This is largely because illegitimacy has become almost the norm. In the speaker's home state, somewhere north of the Mason-Dixon line, 20 percent of white children, 64 percent of Hispanic children, and 77 percent of African-American children are born out of wedlock. The speaker suggested that children raised without love become adults filled with

hate. He challenged organized medicine to do something about it. He suggested that we, as physicians, have the duty to educate people how to raise their children, how to fill their lives with love.

Quixotic? Perhaps. But driving home from the medical society meeting that night, I had two thoughts. First, one should definitely try to attend the county medical society's meetings, for you'll never know what you'll learn or re-learn. Second, I formed the opinion that the speaker's basic message was hardly new. Indeed, I recalled in 1920 a lead article in *The Journal of the American Medical Association* was entitled “The Rights of the Child.” Its author, by no coincidence, was our own State Health Officer and a former SCMA president—Dr. James Adams Hayne.<sup>1</sup>

After reviewing the evolution of our ideas about “the rights of man” and “the rights of woman,” the eloquent Dr. Hayne asked

what of the child? Who has championed its right? Summoned against its will and without its consent into this world of trouble, pain, sickness, and finally death, what Rousseau or Voltaire shall sound the tocsin and call on the infant “mewling and puking in the nurse's arms,” to demand that its own mother shall give it the lactic fluid that is its primal right.... Who shall demand for them the right to believe in fairies, the right to sunshine and to flowers, to the fresh air, and to green fields, to all that makes up the paradise of childhood. Who shall led another Children's Crusade for these, their ancient rights?

He elaborated on the following rights in order of priority:

- The right to be conceived.
- The right to be born after conception.
- The right to have healthy parents.
- The right that “its mother shall be so cared for by the state that neither through poverty nor ignorance shall she bring it into the world in unhygienic surroundings...”
- The right to “have its own mother's milk

until it is old enough to be weaned.”

- The right, “guaranteed by the state, that every baby born shall have an equal chance with other babies to develop into the normal healthy child.”
- “The right that, when it reaches school age, it shall have the best that the resources of any government can command.”

Hayne, it seems, was in some respects well ahead of his time.

More recently, the American Academy of Pediatrics has formulated a similar list of desiderata for children. Some refinements to Hayne’s list of rights include the following:

- The right to immunizations.
- The right to good nutrition.
- The right to education about health and the health care system.
- The right to a safe environment.
- The right to adequate assistance for any handicaps.
- The right to live in a family setting “with an adequate income to provide basic needs to insure physical and intellectual health.”
- The right to be free of environmental contamination.
- The right to live in a society that recognizes the special needs of young people.

And to this list, one might easily add the right to be wanted and the right to be loved.

Why bother with organized medicine? Perhaps the most basic reason is that it takes collective action not only to preserve and strengthen our own traditions but also to improve society. It should not escape our notice that medicine is being re-defined. It has even been suggested that primary care medicine is almost an anachronism—that most functions of the front line physician can in fact be filled by others.<sup>2</sup> But let us not forget that the word “doctor” means “teacher” and that we do, indeed, have the right and the duty, the responsibility and the privilege to teach our patients how to live, learn, and love. And let us not forget the enthusiasm with which Dr. Hayne ended his address: “If the rights I have tried to indicate are given the child, the future of the race is assured.”

—CSB

#### REFERENCES

1. Hayne JA: The rights of the child. *JAMA* 75: 143-145, 1920.
2. Starfield B: Is primary care essential? *Lancet* 344: 1129-1133, 1994.



# Gray Matter

*“Matters of Interest  
to South Carolina  
Physicians.”*

Thornton & Thorne give the medical community something to think about this month.

## **A TIME FOR ACTION**

### **UPDATE ON DISABILITY INSURANCE**

Last month we wrote about the crisis in the disability insurance marketplace. This situation is caused by the significant losses experienced by companies on their disability products.

The individual disability marketplace is dominated by less than a dozen companies. The top half of these companies probably write over 60% of all individual policies.

UNUM and Provident Life and Accident rank among the very top companies. Both recently made important announcements which will greatly impact the disability insurance policy you will be able to buy in the future.

The most startling announcement was from UNUM which announced that it will cease offering *non-cancelable* disability

policies in the United States in 1995 (premiums for non-cancelable policies cannot change prior to the insured's age 65).

Provident announced the introduction of *guaranteed renewable* disability policies for professionals (premiums for guaranteed renewable policies can be changed as experience dictates).

UNUM and Provident are market leaders. When they take bold steps of this kind, it means all disability companies will be making far-reaching changes in the immediate future.

Physicians will be more adversely affected by the changes than any other class of insureds:

- Certain medical specialties have already been downgraded by some companies. The others will follow suit. Policies for these specialists will cost more and definitions will not be as favorable.
- Premiums for physicians will increase significantly across the board. Companies will shorten benefit periods and lengthen elimination periods. Benefit periods for mental or emotional claims will be restricted.
- Both health and financial underwriting will get tougher. The amount of benefit the companies will issue will be reduced.

The time may soon come when you cannot buy a policy with a guaranteed premium. You can currently but you must act quickly.

**SCMA members get a 25% discount on disability policies from Connecticut Mutual. This contract has not changed since late 1992 but change is imminent and could occur at any time.**

## **RECOMMENDATION**

**We recommend that you purchase as much individual, non-cancelable insurance as you can get before Connecticut Mutual announces changes. Once you are issued a policy, the premium cannot be changed prior to your age 65 nor can any restriction be put on the policy.**

**Premiums will never again be as low as they are today. Policy language will never again be as good as it is today.**

**If you are ever again going to acquire disability insurance, DO IT NOW.**

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Views expressed herein are those of the authors only and in no way represent the SCMA. We do not give tax advice. Only your attorney and accountant are qualified to do so.



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# Letters to the Editor

To the Editor:

In the recent excellent article by Ervin,<sup>1</sup> there was a statement that appears to be misleading. The statement is that "testing simultaneously for many individual parameters increases the chances of receiving an 'abnormal' result by  $(0.95)^n$ th power when  $n$  tests are run." However, as  $n$  becomes larger,  $(0.95)^n$  becomes progressively smaller rather than larger. A more accurate statement would be that for  $n$  independent tests, in which the "normal range" has been arbitrarily defined as enclosing 95 percent of the values obtained from healthy subjects, the probability that at least one of these  $n$  parameters will be outside its normal range is equal to  $1-(0.95)^n$ .

For a single test, the probability is  $1-(0.95)^1=0.05$ . That is, by definition of the normal range for this test, a normal subject has a five percent chance of having a value outside of the normal range. A more enlightening example would be to look at the case of the multiphasic metabolic panel commonly referred to as an SMA-12. The probability that a completely healthy individual will have at least one measured parameter on the SMA-12 outside the normal range is  $1-(0.95)^{12}=0.46$ . Therefore, for this common test there is approximately an even chance that not all the parameters will be normal. Not all clinicians are aware of this statistical consequence. The task of the clinician is to be able to recognize when a variance is significant and to be aware of patterns of variances. A fasting blood glucose of 125 (with a normal range of 80-120) may well be a statistical variation while one of 250 is indicative of abnormal glucose metabolism. In the appropriate clinical setting, a SMA-12 having a mildly elevated glucose and alkaline phosphatase suggests the possibility of diabetes with fatty infiltration of the liver. Ervin's discussion of the pitfalls of ordering and interpreting diagnostic tests without an appreciation of their inherent limitations for a defined population is well taken.

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## REFERENCE

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To the Editor:

There has been a recent important South Carolina malpractice suit found in favor of the defendant physician; the case concerned the issue of cephalosporin cross reactivity with penicillin. The defendant used Ceclor in a patient who had a possible history of penicillin reaction/allergy. The patient developed a Ceclor immune complex reaction.

The notion that penicillins cross react with all cephalosporins is across all medicine, but is generally false. Some textbooks of Medicine, Pharmacy and Dermatology contain specific contraindication language about using cephalosporins in penicillin sensitive patients. The 1994 Physician Desk Reference is softer – "In penicillin-sensitive patients, cephalosporin antibiotics should be administered cautiously. There is clinical and laboratory evidence of partial cross-allergenicity of the penicillins and the cephalosporins and there are instances in which patients have had reactions, including anaphylaxis to both drug classes." (This language is legalese and not much help in our clinical practices.)

The first order cephalosporins often DO cross react with penicillins since the antigenicity for both is a similar Beta-lactam ring. Also, the early preparations of cephalosporin were contaminated with "real" penicillin (both were originally made in the same facility from the respective molds which are similar). And so the word was passed and has become ingrained in our medical culture and literature (myths die hard, even in medicine).

But the second and third order cephalosporins are different. First of all there is no



longer any penicillin contamination. But more importantly their antigenicity is in the side chains (NOT the Beta-lactam ring), so there is no (to little) cross reactivity in the scientific sense. Finally, penicillin sensitive patients react more to all other drugs than the general population. The expected "natural" reaction rate to cephalosporins would be three to seven percent (the range of the reported cross-reaction rates so, these cross-reactions are probably not cross-reactions at all). Now of course patients do react to cephalosporins, even second and third order agents, but these reactions are USUALLY UNRELATED TO PENICILLIN SENSITIVITY, although both may coincidentally exist in the same patient

One further point. We have caused ourselves problems with the legal profession in that we use the word "allergy" too loosely. We should stop using the word allergy for non-immune reactions and side-effects. I would prefer that we restrict the word allergy to IgE reactions (most penicillin/cephalosporin reactions are IgG-immune complex reactions or simple side effects).

So how should we proceed with these penicillin sensitive patients? I suggest the patient with penicillin sensitivity be given second or third order cephalosporins straight out if the reaction was a side effect, papular rash, large local reaction or mild fever/arthralgias. With classical allergy reactions (hives, angioedema, asthma or anaphylaxis), I would skin test and if positive for penicillin allergy, then the patient should be referred to an allergist for cephalosporin skin testing because the penicillin/cephalosporin class of drugs are often still the drugs of choice for several infections. With IgG-immune complex reactions I would use cephalosporins only after documented informed consent (make the point that these immune reactions can occur to any drug, at any time, in any patient, and are not related to cross reactions).

For further reading see: Chapter 7 Allergic Reactions to Drugs and Biological Agents, JAMA 268:2845, 1992. Immediate hypersensitivity reactions to beta-lactam antibiotics,

Ann Int Med 107: 204, 1987. Cross sensitivity to cephalosporins in patients allergic to penicillin, *Pediatr Infect Dis*: 557, 1986.

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(Supported by the SC Allergy Society

Brian S. Dantzler, M. D., President)

To the Editor:

While I applaud the efforts of Dr. Holgate<sup>1</sup> to bring modern therapeutic interventional techniques to the attention of your readers, I have several concerns regarding his prescription for the use of angioplasty in the management of occlusive cerebrovascular disease. As the author indicates, the performance of transluminal angioplasty is certainly feasible for stenotic and occlusive disease of the brachio-cephalic, subclavian, vertebral, and carotid arteries. However, the indications, the safety, and the long-term efficacy remain a matter of considerable debate. The mere presence of a subclavian or vertebral artery stenosis discovered angiographically alone is rarely a reason for intervention, either radiologically or surgically.

A clearer description of the clinical indications would be helpful. Dr. Holgate may be aware of randomized prospective controlled trials comparing the efficacy of surgical intervention for cerebrovascular occlusive disease versus angioplasty, but I am not. In fact, only recently has surgical intervention (specifically carotid endarterectomy) been shown to have benefit over a non-interventional therapy for selected patients.<sup>2</sup> As experience with the extracranial to intracranial bypass trial illustrated,<sup>3</sup> the successful performance of a procedure does not necessarily translate into an improved outcome for patients.

Dr. Holgate appears to have a unique understanding of the effect of angioplasty on diseased atherosclerotic vessels. He suggests that "angioplasty dilates the stenosis and flattens the diseased endothelium." More accurately, the angioplasty fractures and disrupts the atherosclerotic plaque leaving an irregular

surface on which fibrin and platelets aggregate. Eventually over time this remodels to allow maintenance of an increased lumen diameter in the peripheral vessels. However, since the results of transluminal angioplasty have widely disparate results in various arterial locations, we must be cautious in transposing such results to the cerebrovascular vessels. Furthermore, his supposition that "since angioplasty does not remove the endothelium, regrowth will prove superior" reflects a misperception regarding current concepts of vessel wall biology.

The ultimate goal of cerebrovascular intervention for occlusive disease is really clinical: a reduction in both cerebrovascular symptoms and long-term stroke risk with minimal morbidity. We must be wary about

recommending procedures that are in search of an indication to our patients simply because they are technically feasible.

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# On the Cover:

## WARING HISTORICAL LIBRARY CENTENNIAL

The quaint, octagonal, brick building featured on this month's cover, the Waring Historical Library of the Medical University of South Carolina, was built in 1894 at a cost of \$7,500. At that time the area was the site of the Porter Academy, a boys' school founded after the Civil War to educate boys left orphaned and destitute by the war. The library building was the gift of the Reverend Charles Frederick Hoffman, D. D., Rector of All Angels Church in New York City, for whom it was named. The Hoffman Library served Porter until 1965.

In 1966, after some discussion of razing the building to make a parking lot had been squelched by cooler heads, the library was turned over to Dr. Joseph I. Waring to house his "musty old books." These books were mostly the "once proud library of the Medical Society of South Carolina" that Dr. Waring had rescued from improper storage and was attempting to save. In 1969 the library was renamed The Waring Historical Library, and today serves not only the Medical University,

but also scholars from all over the world who are interested in Medical History.

The collections have grown over the years. Although the library has many fine classics in the history of medicine, the major emphasis in collecting today is southern medicine with a special interest, of course, in South Carolina.

The Waring staff is involved in acquisition and preservation of collections; assisting researchers; and original research. If you would like to assist in the preservation of South Carolina's medical heritage, you may join the Waring Library Society, a friends of the library group, with the coupon below, and consider the Waring as a possible repository for important books, papers, and artifacts relating to medical history.

The Waring Library is open weekdays from 8:30 to 5:00 pm. Researchers and browsers are welcome.

Betty Newsom  
The Waring Historical Library

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# Alliance Page

## SCMAA PHYSICIANS FAMILY SUPPORT

All families experience difficulties from time to time and medical families are certainly no exception. In fact, along with the usual array of difficulties, medical families sometimes encounter problems unique to their circumstances. Aware that a support network can be invaluable in helping families cope, the South Carolina Medical Association and its Alliance provide help through their Physicians Family Support Committee.

Christine Hepfer (Mrs. Thomas W.) serves as chairperson of this committee. She sends notes of support to the physician members of the SCMA and their families. These can be notes of congratulations for passing "the boards" or for birthdays. It can also be notes of sympathy and support to the family with the loss of a family member. If you would like us to remember, congratulate, or support a member of his/her family please contact her at 2810 Lillington Drive, Sumter, SC 29150-2213.

Barbara Clark (Mrs. Terence M.) is the co-chairperson and serves as the liaison between the Alliance and the SCMA Physicians Advocacy and Assistance Committee. A key purpose of the SCMAA Physicians Family Support Committee is to work closely with the SCMA committee to insure that support, assistance and encouragement are available to physicians' families who are experiencing problems and concerns related to some type of impairment (chemical dependency and addiction, mental illness, grief, aging, long-term illness, marital problems, malpractice, problems with children, etc.)

Under the extremely able leadership of Kaye Borgstedt, the subcommittee established the following goals which continue to be the focus for 1994-1995: (1) maintain and strengthen membership in the four regional "Caduceus-Alanon" groups; (2) encourage more spouses to participate with the physicians in their quarterly meetings; (3) continue educational efforts to raise awareness and make resources known; (4) continue to meet with and participate with the physicians' group; and (5) strengthen the organization and structure of the spouse regional team groups.

In an effort to raise awareness of the widespread but often "hidden" problem of chemical dependency, the committee provides speakers for medical society and/or alliance meetings. The speaker will share, on a very personal level, experiences and anecdotes as well as ways in which the problem can not only be overcome but serve as a powerful, positive growth opportunity. This service is provided as a means to encourage families who suspect some type of impairment to seek help.

If you or a family member are struggling with a painful or difficult problem or concern, have questions or need information, would like to arrange for a speaker to share at a meeting, or would like to be a part of this group and help others, please contact Cathy Boland at SCMA Headquarters at 1-800-327-1021, ext. 232, or Barbara Clark at 1-803-654-5680. We have all appreciated the help and support of others from time to time. Our committee stands ready to be of whatever help we can to physicians and their families. Sometimes just a "listening ear" or a brief inquiry can be a helpful first step. We want to make that first step an easy and comfortable one.

Christine Hepfer, Chairperson

Barbara Clark, Co-Chairperson

# INDEX TO VOLUME 90, 1994

## AUTHORS

- Abramson, Ruth K., Ph. D. ...407  
Afrin, Lawrence B., M. D. ...231  
Bailey, Byron N., M. D. ...13  
Bachman, David, M. D. ...373, 553  
Baskins, Judith Pinner, R. N. ...586  
Bates, William, M. D. ...360  
Bennett, Michelle ... 105  
Bissada, Nabil K., M. D. ...209, 220  
Boone, William R., Ph. D. ...360  
Bowers, Betty K., M. N. ...360  
Brannon, William L., M. D. ...399, 402  
Brown, Peggy ...318  
Bucci, Michael N., M. D. ...270  
Camp, Mary, R. V. T. ...318  
Cardona, Fé A., M. D., M. P. H. ...285  
Carter, Richard, M., M. D. ...517  
Carter, Timothy D., M. D. ...310  
Christie, Scott D., M. D. ...97  
Cooke, James, M. D. ...450  
Cooner, William H., M. D. ...210  
Cornman, Carol B., R. N., P. A. ...51, 404  
Courville, Timothy, M. D. ...491  
Crane, Martin M., Ph. D. ...360  
Davis, Dorothy R., B. A. ...51  
Davis, Jacqueline, R. V. T. ...318  
Davis, Paul T., M. D. ...479  
Deci, Paul A., M. D. ...97, 101  
Donald, Alexander G., M. D. ...377  
Ector, Walton L., M. D. ...355  
Egbert, John, M. D., Ph.D. ...586  
Eleazer, G. Paul, M. D. ...404, 586  
Ervin, Daniel E., M. D. ...483  
Ervin, F. Richard, M. D. ...58  
Evans, O. Norman, M. D. ...113  
Faulkner, Larry R., M. D. ...113  
Fenwick, Jeffrey R., M. D. ... 513  
Fischinger, Peter, M.D., Ph. D. ...242  
Flandry, Robert E., Jr., M. D. ...11  
Fogartie, J.E., Jr., M. D, R. V. T. ...318  
Frank, Elaine M., Ph. D. ...417  
Fravel, William J., M. D. ...499  
Funcik, Thomas, M. D. ...507  
Garovich, Michael C., M. D. ...314  
Giles, William C., M. D. ...479  
Going, Jacquelyn A., M. D. ...483  
Goldman, Charles R., M. D. ...105  
Harrell, Laurie R., M. D. ...222  
Hellstrom, Michael, M. D. ...505  
Herman, Kerri, B. S. ...373  
Hicks, Patricia W., R. N. C. ...105  
Hiers, Thomas G., M. D. ...97  
Hiott, D. Walter, M. D. ...97  
Hobson, J.R., Jr., M. D. ...318  
Hochman, Marcelo, M. D. ...448, 450, 455  
Hodo, Gary L., Ph. D. ...113  
Hoffman, Brenda J., M. D. ...281  
Holgate, Richard C., M. D. ...23  
Holloway, W.O., M. D. ...318  
Horton, Joseph, M. D. ...450  
Howard, Gene, M. D. ...450  
Huang, Youjie, M. D., M. P. H. ...51, 404  
Hunt, Steven, M. S. W. ...373  
Isenhower, W. David, Jr., M. D. ...517  
Jenrette, Joseph M., III, M. D. ...220, 450  
Johnson, C. David, M. D. ...586  
Johnson, Jane E., M. T. ...360  
Johnson, John K., M. D. ...16  
Johnson, N. Peter, Ph.D. ...601  
Kemper, Karen A., Ph. D. ...263  
Key, Janice D., M. D. ...355  
Kirkland, T.A., M. D. ...217  
Kitch, Russell, D., M. D. ...513  
Kurent, Jerome E., M. D. ...310  
Lang, Pearson G., M. D. ...455  
Lathem, J.E., M. D. ...217  
Leggett, Richard P., M. D. ...105  
Leung, Alexander K. C., M. B., B. S. ...592  
Lewis, Linda, M. S. N. ...373  
Macera, Carolina A., Ph. D. ...51, 404  
Malone, Michael J., M. D. ...539  
Mathers, Michael S., M. D. ...592  
McLeod-Bryant, Stephen, M. D. ...101  
Meador, Kimford J., M. D. ...543  
Meeks, Alison, R. N. ...373  
Milling, Robert N., M. D. ...93  
Mintzer, Jacobo E., M. D. ...373, 553  
Mohler, James L., M. D. ...214  
Morris, M. Dawn, B. S. N. ...360  
Morrison, Edward C., M. D. ...327  
Neff, Linda, B. S. ...51  
Nelson, Robert P., Jr., M. D. ...237  
Newburg, Julie A., M. D. ...485  
Osguthorpe, David, M. D. ...448, 450  
Pacult, Artur, M. D. ...5, 6, 309  
Parrott, Larry H., M. D. ...56  
Parsons, Gregory S., M. D. ...501  
Patel, Sunil, M. D. ...450  
Pennypacker, Leslye, M. D. ...373  
Phillips, John G., M. D. ...495  
Philyaw, Doris C., B. S. N., R. N. ...270  
Pitner, Janet, Pharm. D. ...373  
Plyler, John W., M. D. ...330  
Poland, Robert M., M. A, M. A., CCC-A. ...485  
Price, Thomas M., M. D. ...360  
Riester, Jane W., M. D. ...505  
Roberts, Walter J., Jr., M. D. ...31, 341  
Robson, William Lane M., M. D. ...592  
Sampson, Royce, M. S. N. ...373  
Santos, Alberto B., M. D. ...97, 101  
Sargent, Roger G., Ph. D. ...263  
Schlidl, Robert A., M. D. ...281  
Schulken, Ellen, Ph. D. ...263  
Scott, William K., M. S. P. H. ...51, 404  
Simpson, William, M. D. ...373  
Smith, Charles E., M. D., D. M. D. ...495  
Stands, Benjamin O., M. D. ...601  
Still, Charles N., M. D. ...397, 533, 560  
Strothers, Harry S., III, M. D. ...601  
Stuart, Robert K., M. D. ...231

Thomas, Neal J., M. D. ....355  
 Waters, Robert C., M. D. ...485  
 Watson, Theodore A., M. D. ...499  
 Weber, Peter C., M. D. ...450, 517  
 White, James F., M. D. ....507  
 Wickwar, Hardy ...69  
 Williams, Arthur V., M. D. ...  
 Wilson, Brian D., M. D. ....501  
 Wilson, Lisa, M. D. ....586  
 Wohlreich, George M., M. D. ....373  
 Wohlreich, Madeline M., M. D. ...120  
 Wolin, Michael Jay, M.D. ....534  
 Wright, Lore K., Ph. D., R. N. C. S. ....424

## ORIGINAL SCIENTIFIC ARTICLES

Bicycle-Related Head Injuries in South Carolina Children: What Can We Do to Prevent Them?—Neal J. Thomas, M. D., Janice D. Key, M. D., Walton L. Ector, M. D. ....355  
 Dietary Behaviors of South Carolina Adolescents—Roger G. Sargent, Ph. D., Karen A. Kemper, Ph. D., Ellen Schulken, Ph. D. ....263  
 Incidence of Astrocytoma in Anderson County: A Three Year Analysis—Michael N. Bucci, M. D., Doris C. Philyaw, B. S. N., R. N. ....270  
 Infectious Esophagitis and the Primary Care Physician—Robert A. Schlidt, M. D., Brenda J. Hoffman, M. D. ....281  
 In Vitro Fertilization and Embryo Transfer (IVF-ET) Comes to the Upstate—William R. Boone, Ph. D., Jane E. Johnson, M. T., Thomas M. Price, M. D., Betty K. Bowers, M. N., M. Dawn Morris, B. S. N., Martin M. Crane, Ph. D., William Bates, M. D. ....360  
 Kidney Function in Congestive Heart Failure—Arthur V. Williams, M. D. ....579  
 Managed Care for the Frail Elderly: The Pace Project—G. Paul Eleazer, M. D., Judith Pinner Baskins, R. N., John Egbert, M. D., Ph. D., C. David Johnson, M. D., Lisa Wilson, M. D. ....586  
 (A) New Approach for the Management of Acute Psychiatric Disorders in Elderly Demented Patients—Jacob E. Mintzer, M. D., Linda Lewis, M. S. N., Leslye Pennypacker, M. D., Janet Pitner, Pharm. D., William Simpson, M. D., David Bachman, M. D., George M. Wohlreich, M. D., Alison Meeks, R. N., Steven Hunt, M. S. W., Kerri Herman, B. S., Royce Sampson, M. S. N. ....373  
 Renal Colic due to Henoch-Schönlein Purpura—William Lane M. Robson, M. D., Alexander K. C. Leung, M. B., B. S., Michael S. Mathers, M. D. ....592  
 Survival of Alzheimer's Disease Patients with Regard to Pattern of Care in South Carolina, 1988-1993—Youjie Huang, M. D., M. P. H., Caroline A. Macera, Ph. D., Carol B. Cornman, B. S., P. A., Dorothy R. Davis, B. A., William K. Scott, M. S. P. H., Linda Neff, B. S. ....51

## SPECIAL ARTICLES

Milestones in the History of Mental Retardation—Fé A. Cardona, M. D., M. P. H. ....285  
 Neighborcare—Hardy Wickwar. ....69  
 Rationalizing Decisions for Diagnostic Testing—F. Richard Ervin, M. D. ....58

Report of the SCMA Delegation to the AMA Annual Meeting—Walter J. Roberts, Jr., M. D. ... 341  
 Report of the SCMA Delegation to the AMA Interim Meeting—Walter J. Roberts, Jr., M. D. ....31  
 Roderick Macdonald, M. D.: A Tribute—Alexander G. Donald, M. D. ....377  
 You can do Something about Adolescent Substance Use and Abuse: A Practitioner's Guide to Adolescent Alcohol and other Drug Prevention—N. Peter Johnson, Ph. D., Benjamin O. Stands, M. D., Harry S. Strothers, III, M. D. ....601

## SPECIAL ISSUES

### Alzheimer's Disease: Part 1

Guest Editor: Charles N. Still, M. D.  
 Introduction—Charles N. Still, M. D. ....397  
 Alois Alzheimer (1864-1915) I. Contributions to Neurology and Psychiatry—William L. Brannon, M. D. ....399  
 Alois Alzheimer (1864-1915) II. Dementia Before and After Alzheimer: Brief History—William L. Brannon, M. D. ....402  
 Epidemiology of Alzheimer's Disease—Caroline A. Macera, Ph. D., Youjie Huang, M. D., M. P. H., G. Paul Eleazer, M. D., William K. Scott, M. S. P. H., Carol B. Cornman, R. N., P. A. ....404  
 Genetics of Alzheimer's Disease—Ruth K. Abramson, Ph. D. ....407  
 Effect of Alzheimer's Disease on Communication Function—Elaine M. Frank, Ph. D. ....417  
 Alzheimer's Disease and Caregiver Stress—Lore K. Wright, Ph. D., R. N. C. S. ....424

### Alzheimer's Disease: Part 2

Guest Editor: Charles N. Still, M. D.  
 Introduction—Charles N. Still, M. D. ....533  
 Alzheimer's Disease and the Eye—Mitchell Jay Wolin, M. D. ....534  
 Multi-Infarct Dementia (MID)—Michael J. Malone, M. D. ....539  
 Treatment Strategies for the Cognitive Deficits of Alzheimer's Disease—Kimford J. Meador, M. D. ....543  
 New Approaches in the Treatment of Alzheimer's Disease—Jacob E. Mintzer, M. D., David Bachman, M. D. ... 553  
 Aluminum Neurotoxicity and Alzheimer's Disease—Charles N. Still, M. D. ....560

### Update on Otolaryngology-Head and Neck Surgery

Guest Editors: M. Hochman, M. D.  
 J. David Osguthorpe, M.D.  
 Introduction—M. Hochman, M. D., David Osguthorpe, M. D. ....448  
 Multi-disciplinary Approach to Cranial Base Tumors—J. David Osguthorpe, M. D., Sunil Patel, M.D., Marcelo Hochman, M. D., Gene Howard, M. D., Joseph Jenrette, M. D., James Cooke, M. D., Joseph Horton, M. D., Peter Weber, M. D. ....450  
 The Management of Non-melanoma Skin Cancer of the Head and Neck—Pearon G. Lang, M. D., Marcelo Hochman, M. D. ....455  
 Comprehensive Care of the Cleft Lip and Palate



Patient—William C. Giles, M. D., Paul T. Davis, M. D. ... 479  
 Otitis Media: When are Tubes and/or Adenoidectomy Needed?—Jacquelyn A. Going, M. D., Daniel M. Ervin, M. D. ...483  
 Hearing Aids: Selection, Parameters, & Limitations—Robert M. Poland, M. A., M. A., CCC-A, Robert C. Waters, M. D., Julie A. Newburg, M. D. ...485  
 Cochlear Implants—Timothy Courville, M. D. ...491  
 Snoring and Sleep Apnea: Evaluation and Treatment—John G. Phillips, M. D., Charles E. Smith, M. D., D. M. D. ...495  
 Hay Fever: Pharmacotherapy or Immunotherapy?—William J. Fravel, M. D., Theodore A. Watson, M. D. ...499  
 If Surgery is the Last Option for Sinusitis, What is the First?—Gregory S. Parsons, M. D., Brian C. Wilson, M. D. ...501  
 Common Voice Disorders—Jane W. Riester, M. D., Michael Hellstrom, M. D. ...505  
 Options in Cosmetic Rehabilitation of the Aging Face—Thomas Funcik, M. D., Russell D. Kitch, M. D. ...507  
 Evaluation of the Neck Mass—Jeffrey R. Fenwick, M. D., Russell D. Kitch, M. D. ...513  
 The Evaluation and Diagnosis of the Dizzy Patient—W. David Isenhower, Jr., M. D., Richard M. Carter, M. D., Peter C. Weber, M. D. ...517

### **Cerebrovascular Diseases Part 1**

Guest Editor: Artur Pacult, M. D.  
 Introduction—Artur Pacult, M.D. ...5  
 Cerebral Aneurysms, Subarachnoid Hemorrhage: An Overview of Incidence, Location, Presentation, Diagnosis and Therapy—Artur Pacult, M. D. ...6  
 Inflammatory Intracranial Aneurysms—Robert E. Flandry, M. D. ...11  
 Cerebral Arteriovenous Malformations—Byron N. Bailey, M. D. ...13  
 Vascular Anomalies in the Pediatric Age Group—John K. Johnson, M. D. ...16  
 Interventional Techniques in the Management of Disorders of the Brain: Current Status—Richard C. Holgate, M. D. ...23

### **Cerebrovascular Diseases Part 2**

Guest Editor: Artur Pacult, M. D.  
 Introduction—Artur Pacult, M. D. ...309  
 Clinical Syndromes of Cerebral Ischemia—Timothy D. Carter, M. D., Jerome E. Kurent, M. D. ...309  
 Medical Imaging of Cerebral Ischemia—Michael C. Garovich, M. D. ...314  
 Color Flow Duplex Scanning in Evaluation of Carotid Artery Atherosclerotic Occlusive Disease—J. E. Fogartie, Jr., M. D., R. V. T., W. O. Holloway, M. D., J. R. Hobson, Jr., M. D., Jacqueline Davis, R. V. T., Mary Camp, R. V. T., Peggy Brown ...318  
 The Surgery of Extracranial Vascular Occlusive Disease—Edward C. Morrison, M. D. ...327  
 Medical Management of Ischemic Cerebrovascular Disease—John W. Plyler, M. D. ...330

### **Prostate Cancer**

Guest Editor: Nabil K. Bissada, M. D.  
 Introduction—Nabil K. Bissada, M.D. ...209  
 A Practical Approach to Prostate Cancer Detection in 1994—William H. Cooner, M. D. ...210  
 Diagnosis and Staging of Prostatic Carcinoma—James L. Mohler, M. D. ...214  
 The Role of Transrectal Ultrasound (TRUS) in the Evaluation of Cancer of the Prostate— T. A. Kirkland, M. D., J. E. Lathem, M. D. ...217  
 Introduction to Management of Prostate Cancer—Nabil K. Bissada, M. D. ...220  
 Surgical Management of Prostate Cancer—Nabil K. Bissada, M. D. ...220  
 Prostate Cancer: The Role of Radiation Therapy—Joseph M. Jenrette, III, M. D., Laurie R. Harrell, M. D. ...222  
 Medical Therapy of Prostate Cancer—Lawrence B. Afrin, M. D., Robert K. Stuart, M. D. ...231  
 Diagnosis and Treatment of Erectile Dysfunction and its Relationship to Prostatic Carcinoma—Robert P. Nelson, Jr., M. D. ...237  
 The Evolving Perspective of Prostate Cancer: A Question of Natural History and Current Treatment Trends—Peter J. Fischinger, M. D., Ph. D. ...242

### **Current Issues in Psychiatry, Part 2**

Guest Editor: Robert N. Milling, M. D.  
 Introduction and Cover Story—Robert N. Milling M. D. ...93  
 Intensive Outpatient Intervention Reduces Need for Inpatient Care of Chronically Psychotic Patients—Albert B. Santos, M. D., Paul A. Deci, M. D., Scott D. Christie, M. D., Thomas G. Hiers, M. D., Walter Hiott, M. D. ...97  
 MUSC's Rural Mental Health Services Initiatives—Paul A. Deci, M. D., Alberto B. Santos, M. D., Stephen McLeod-Bryant, M. D. ...101  
 Treating Patients with Mental Illness and Chemical Dependency: An Intensive Outpatient Program—Charles R. Goldman, M. D., Patricia W. Hicks, R. N. C., Michelle Bennett, Richard P. Leggett, M. D. ...105  
 The Impact of HIV Positive Patients on the South Carolina Department of Mental Health—Gary L. Hodo, Ph. D., O. Norman Evans, M. D., Larry R. Faulkner, M. D. ...113  
 Postpartum Emotional Illness: Recognition and Management in Primary Care—Madeline M. Wohlreich, M. D. ... 120

### **EDITORIALS**

A Matter of Time—Charles S. Bryan, M. D. ...74  
 An Overview—Artur Pacult, M. D. ...35  
 Counseling the Patient with Prostate Cancer—Nabil K. Bissada, M. D. ... 253  
 Deserving Our Remembrance—Charles S. Bryan, M. D. ...383  
 Editorial—Marcelo Hochman, M. D. ...523  
 Of Ideals and Heroes—Charles S. Bryan, M. D. ...221  
 (The) Magnitude of the Problem—Artur Pacult, M. D. ...339  
 ("The) Rights of the Child" Revisited—Charles S. Bryan, M. D. ...613

Third World Cardiology: Back to Basics—N. B. Baroody, M. D. ...611  
Whither Academic Health Centers?—Charles S. Bryan, M. D. ...295

#### FEATURES

Alliance Page ...43, 83, 133, 198, 255, 298, 347, 389, 436, 524, 571, 621  
Letters to the Editor ...78, 617  
On The Cover ...37, 80, 93, 197, 254, 297, 345, 386, 435, 523, 569, 620  
President's Page ...3, 49, 91, 141, 207, 261, 307, 353, 395, 445, 531, 577

#### ASSOCIATION

CME Calendar ...129, 289, 429, 607  
Erratum ...276  
Gray Matter. ...41, 81, 137, 195, 249, 299, 349, 387, 437, 511, 565, 615  
Index to Volume 90 ...622  
Information for Authors. ... 38  
Physician Recognition Awards ...44, 159, 515, 596  
SCMA Newsletter ...19, 65, 109, 155, 277, 323, 369, 413, 475, 549

#### ONE HUNDRED FORTY-SIXTH ANNUAL MEETING

Introduction ...143  
Schedule of Events ...144  
Delegates and Alternates ...160  
Officer Reports ... 165  
Committee Reports ... 173  
Report of the Executive Vice President ...180  
Report of the Editor ...181  
SCMA MIT Report ... 181  
SCIMER Report ... 182  
SCMA Delegation to the AMA ... 182  
Physicians Care Network ... 183  
Young Physicians Section ...184  
DHEC Report ... 185  
Resolutions ... 190  
AMA Special Guest ... 192  
Exhibitors ...218

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## INDEX TO ADVERTISERS

Anne M. Bowen, C. F. P. ....	596
ASTPHLD.....	596
The Cliffs at Glassy.....	Cover 2
Deweese Island .....	Cover 3
Interim Health Care.....	576
MedCorp .....	575
Medical Protective Company.....	Cover 4
E. G. Todd Physician Search.....	619
Ultimate Auto Collection.....	612
United States Air Force.....	585
United States Air Force Reserve .....	625
Winchester Surgical Supply Company .....	591



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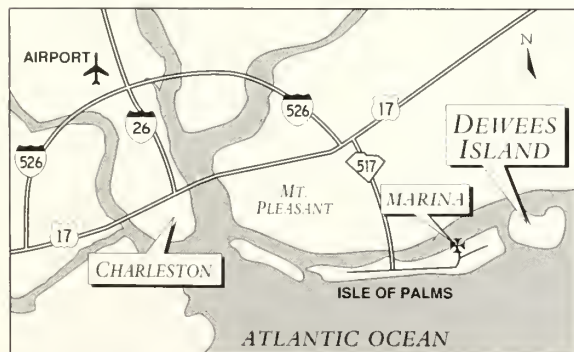
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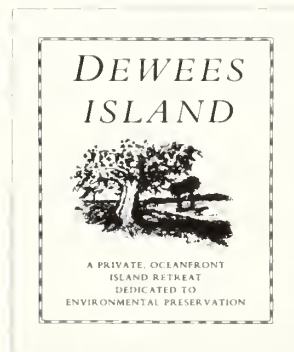
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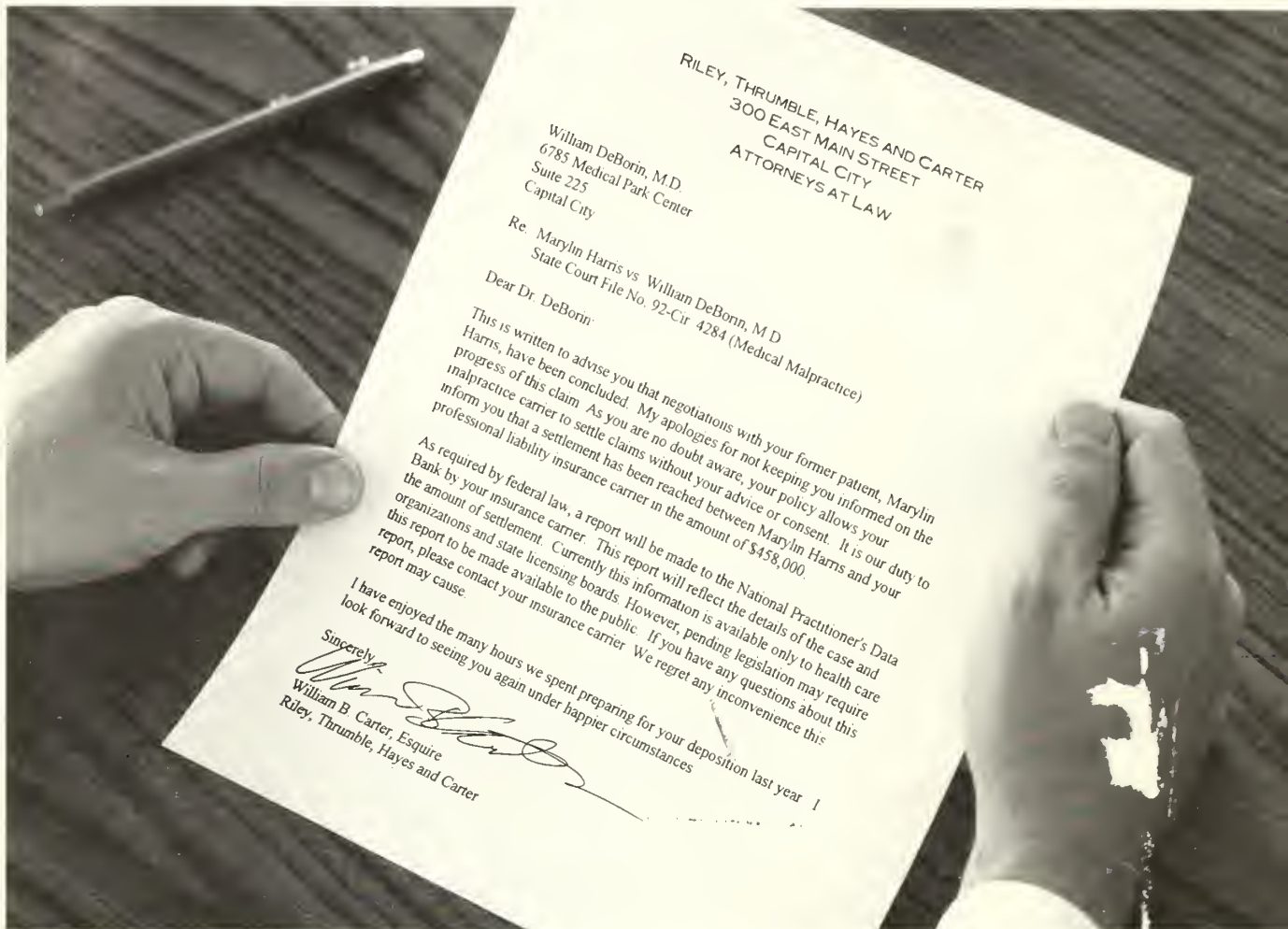
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